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ORIGINAL ARTICLE

A cross-sectional examination of executive function and its associations with grazing in persons with obesity with and without eating disorder features compared to a healthy control group

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Abstract

Purpose The current study aimed to investigate associations between grazing and different facets of executive functioning in persons with obesity with and without significant eating disorder psychopathology, compared to a healthy-weight control group.

Methods Eighty-nine participants (of which 20 had obesity and marked eating disorder symptomatology, 25 had obesity but without marked eating disorder symptoms, and 44 were healthy-weight age- and sex-matched participants; $N = 89$; 66.3% female, age = 28.59 (8.62); 18.18–58.34 years) completed a battery of neuropsychological tests and demographic and eating disorder-related questionnaires. Poisson, Negative Binomial, and Ordinary Least Squares regressions were performed to examine group differences and the associations of grazing with executive functioning within the three groups.

Results Significantly lower inhibitory control and phonemic fluency were observed for the obesity group without ED features compared to healthy-weight controls. Increasing grazing severity was associated with improved performance in inhibitory control in both groups with obesity, and with phonemic fluency in the obesity group with marked eating disorder features.

Conclusion Although there is mounting evidence that specific cognitive domains, especially inhibition, are affected in obesity, evidence of further detrimental effects of eating disorder psychopathology remains mixed; additionally, for persons with obesity, there may be a weak but positive link between executive functioning and grazing behaviour.

Level of evidence III, comparative cross-sectional observational study with a concurrent control group.

Keywords Obesity · Eating disorders · Executive functioning · Grazing · Inhibition · Phonemic fluency

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Introduction

Obesity, defined as abnormal or excessive fat accumulation that presents a risk to health, is a global cause of morbidity and mortality [1]. Eating disorders (ED) are a group of serious illnesses in which people experience severe disturbances in their eating behaviors and related thoughts and emotions [2]. Some EDs (notably binge eating disorder, which is one of the most common EDs [3], and bulimia nervosa) are common co-morbidities of obesity [4], and rates of disordered eating within obesity have been increasing in prevalence over the past decade [5]. Recent neuropsychological research also suggests that increased adiposity is associated with reduced cognitive performance, particularly executive functions (EF) [6]. EF are the mental processes enabling goal formulation, planning, and carrying out these plans effectively [7], and they play a substantial role in regulating eating behavior [8, 9].

A putative mechanism for the association between cognition and obesity is that chronic, low-grade inflammation reduces cognitive functioning [10], with detrimental effects on self-regulatory processes via reduced EF [11]. Reduced metabolism in prefrontal cortical regions which coordinate EF has been observed in obesity [12], and EF have been seen to improve following weight loss in adults with obesity [13]. Further, a recent meta-analysis indicated reductions in obese participants compared to healthy-weight controls across all main EF domains [14]. It is possible that EF deficiencies could predispose individuals to weight gain or to an inability to lose weight successfully [15], or that a bidirectional relationship exists between obesity and EF [6, 14].

Reduced activity in frontostriatal circuits has also been observed in binge-type ED [16] and decreased EF are associated with dysfunction characterising both eating and weight disorders [17, 18]. Some research found lower EF in domains such as planning and decision-making when disordered eating was present within obesity [19, 20]. Cognitive training treatments for obesity and disordered eating have also shown promising results [21–23]. However, the literature to date is not clear regarding EF at the intersection of obesity and ED. While some studies report similar EF performance in participants with obesity with and without an ED [24–26], there is also substantial neurobiological and genetic evidence that binge eating disorder (which is highly prevalent in obesity [27]) represents a distinct phenotype within the obesity spectrum, characterised by elevated impulsivity and compulsivity [28].

Eating patterns in obesity and ED are heterogeneous, and whilst the neuropsychological profile of objective binge eating is the most studied, there is growing interest in other types of eating disturbances, such as grazing. Expert consensus has defined grazing as the unplanned, repetitive eating of small amounts of food (i.e. smaller than would constitute a meal), and/or eating not in response to hunger/satiety sensations [29]. It is relatively common in clinical samples with binge-type ED (67.77% in binge eating disorder and 58.25% in bulimia nervosa) and obesity (33.20% at pre-weight loss treatment, 28.16% at follow-up, and 23.32% in the community) [30]. Grazing rates appear to be especially high at the intersection of obesity and ED [31, 32]. Grazing is considered "compulsive" when a sense of loss of control over eating is a predominant feature or "non-compulsive", when it is better defined by a repetitive, distracted quality [33]. Compulsive grazing has been associated with psychological distress [34], ED symptoms [35], symptoms of food addiction [36], binge-type ED and higher weight [37]. It is also higher in persons with obesity, ED (especially binge eating disorder and bulimia nervosa, but also in other EDs), and markedly, where these overlap [37].

Lifestyle interventions for obesity are limited in effectiveness, and it has been increasingly apparent in recent years that cognitive and psychological factors need to be incorporated into treatment [38]. It is therefore important to examine the cognitive drivers of eating behaviours. As grazing has been placed on the spectrum of compulsive eating [33], it may be associated with failures of self-regulation implicating decreased EF. A recent model [39] suggests that EF influences atypical eating behaviours including grazing, thus contributing to the maintenance of high weight. Furthermore, a recent study in persons with obesity and eating patterns including grazing determined that inhibitory control deficits improved with treatment specifically targeting this EF [40]. Currently, however, there is very little information on the neurocognitive correlates of grazing.

Therefore, this study aimed to investigate associations between grazing severity and any differences in EF present in persons with obesity with and without significant ED psychopathology, compared to a healthy-weight control group, while controlling for factors known to influence EF performance [41, 42]. It was hypothesised that (1) the neuropsychological performance of the healthy control group would be better than that of the group with obesity but without significant ED psychopathology, which in turn would display higher performance than the group with obesity and significant ED psychopathology; (2) any decrements in executive functioning found would be negatively associated with grazing, such that decreasing performance would be associated with increasing grazing severity.

Materials and methods

Participants

Ninety participants aged 18–65 years, with BMI in the "healthy" ($18.5 \leq BMI \leq 25$; $n = 45$) or "obese" ($BMI \geq 30$; $n = 45$) range, who had completed ≥ 10 years of education in English were recruited from community and university settings in Sydney, Australia between February 2015 and October 2016. Participants were recruited via online advertisements placed on websites such as Gumtree and Craigslist, and via flyers placed around the university. All participants were screened over the telephone prior to face-to-face participation. Exclusion criteria consisted of history of psychosis/mania, neurological disorders, learning disorders, hearing/visual impairment, regular sedative/stimulant use, substance use difficulties and current participation in weight loss treatment. As reimbursement, community participants received an AUD$20 shopping card, while students received course credit. The study was approved by the University of Sydney Human Research Ethics Committee (2014/936). One healthy-weight participant endorsing significant ED psychopathology was excluded from the control group, leaving $N = 89$ as the final sample. Please see "Appendix B "for the recruitment flow diagram.

Procedure

Participants completed a face-to-face assessment consisting of anthropometric measurement, self-report questionnaires containing demographics and measures of ED psychopathology and mood, followed by neuropsychological tests.

Clinical measures

Anthropometric measurements. BMI (kg/m^2) was calculated using height and weight measured using Tanita Wedderburn BWB-700 scales and stadiometer.

ED features were assessed with the 28-item Eating Disorder Examination-Questionnaire [EDE-Q; 43]. A method for distinguishing participants likely to have an ED in community samples was employed based on Mond et al. [44]: (1) EDE-Q Global Score ≥ 2.3 AND (2) the occurrence of objective binge eating episodes OR exercising for weight/shape reasons at least 1/week.

Grazing severity was assessed with the seven-item Grazing Questionnaire [GQ; 34], which rates grazing frequency on a five-point scale; an additive total score is generated including two factors: repetitive (non-compulsive) grazing (four items; e.g. "Do you eat more or less continuously throughout the day or during extended parts of the day (e.g., all afternoon)?"), and perceived loss of control, or compulsive grazing (three items; e.g. "Have you ever felt that you were unable to stop grazing?"). The two factors were significantly and strongly positively correlated, $r = 0.68$, $p < 0.001$.

Depression severity over the past week was rated on a five-point Likert scale using the seven-item Depression subscale of the Depression Anxiety Stress Scales-21 (DASS-21) [45].

Neuropsychological measures

Full-Scale IQ was estimated using the Test of Premorbid Functioning (TOPF) [46], comprised of a list of 70 words with atypical grapheme to phoneme translations which are read aloud. The raw score consists of the total number of words pronounced correctly, ranging from 0 to 70. This score was converted to a standard score using age norms.

Inhibition was assessed using the Hayling Sentence Completion Test [47]. Part A reflects response initiation: participants completed 15 sentences with an expected word, clearly suggested by the context. Part B reflects the inhibition of a prepotent response: participants had to produce a word that was incongruous in the context of 15 different sentences. Inhibition was operationalised as errors produced in Part B.

Working memory was measured using Wechsler Adult Intelligence Scale-IV Digit Span subtest [48]. Participants were presented with clusters of numbers of increasing length and asked to repeat the numbers in the same order, backward, and in sequential order. The number of correct responses was recorded, ranging from 0 to 48. This raw score was converted to a standard score using age norms.

Planning and organisation were assessed using the Rey Complex Figure Test (RCFT) [49]. Participants produced a freehand copy of an abstract drawing. A raw score was calculated by summing up points obtained for each of the elements of the figure copied correctly, ranging from 0 to 36.

Verbal fluency was measured using the Controlled Oral Word Association Test [50]. Participants generated as many words as possible starting with three letters (F, A, S) in a 1-min interval per letter (phonemic fluency). Participants then generated as many animal names as possible for 1 min (semantic fluency). The number of correct words for each category was recorded.

Visual cognitive flexibility was assessed using the Trail Making Test (TMT) [51]. In Part A, participants draw lines connecting circled numbers in the sequence (i.e., 1–2–3, etc.) as rapidly as possible. In Part B, participants draw lines to connect circled numbers and letters in an alternating numeric-alphabetic sequence (i.e., 1-A-2-B…) as rapidly as possible. To control for psychomotor speed, the B-A time difference was used as the outcome.

Set shifting and perseveration was tested using a computerised version of the Wisconsin Card Sorting Test-64

Card Version (WCST) [52]. Respondents sorted 64 cards according to different principles and had to shift their sorting approach, with the number of perseverative errors recorded.

Statistical plan

Analyses were performed using IBM SPSS Statistics v26. Groups were compared using ANOVAs, Welch tests and χ^2 tests. A two-tailed α of 0.05 was used, and for polynomial and pairwise contrasts Sidak corrections were employed for continuous variables and Bonferroni corrections for categorical variables. For count data, Poisson and Negative Binomial Regression were used, according to data dispersion. For continuous outcomes, Ordinary Least Squares Regression was used; residuals were inspected for normality, without any major departures observed. Unadjusted analyses were first conducted, followed by analyses adjusted for age, sex, education, estimated overall intellectual functioning and depression severity as recommended in prior EF research [42], with no important differences observed. No significant collinearity was detected, and robust standard errors were used for all analyses. Only two participants had missing data; five multiple imputation data sets were generated and analysed, with pooled results compared with original analyses, with no differences found for any of the outcomes.

For cognitive domains displaying between-group differences, the effect of grazing (entered as a continuous variable) on EF was examined within each of the groups, as these significantly varied in terms of their grazing severity.

Power calculations using the G*Power 3 software indicated that with three groups, six covariates, $\alpha = 0.05$, $\beta = 0.80$, and 89 participants, the power for detecting a large effect size ($f = 0.40$) is 0.80.

Results

Sample characteristics

The final sample consisted of 89 participants, 66.3% female, mean age (SD) 28.59 (8.62) years, and mean years of education 16.47 (2.39). Twenty had obesity and significant ED symptoms (OBED), 25 had obesity without significant ED symptoms (OB), and 44 were healthy-weight controls without significant ED symptoms (HC). Full demographic and clinical characteristics can be found in "Appendix A". No significant between-group demographic differences were observed.

Clinical characteristics

BMI, global ED psychopathology, depression and severity of grazing were generally highest in the OBED group, followed by the OB group and then HC (see "Appendix A"). Only the OBED group had an EDE-Q Global Score within the clinical range, and 100% of participants in this group endorsed objective binge episodes, with 65% endorsing at least one episode per week on average. Only two participants endorsed purging (with a frequency lower than the DSM-5 criteria for purging bulimia nervosa). Hence, the OBED group can be conceptualised as being most closely aligned with the "binge eating disorder" category.

Executive functioning

Between-group differences

There was a significant effect of group on inhibition and phonemic fluency in both unadjusted analyses (Table 1) and those adjusted for covariates (Table 2). Across both domains, a linear trend existed, showing a proportional decrease in performance from HC to the OBED to the OB

Table 1 Neuropsychological measures–unadjusted scores

Measure	HC ($n = 44$) M (SD)	OB ($n = 25$) M (SD)	OBED ($n = 20$) M (SD)	*F*/Welch	*df*	*p*	ηp^2	Pairwise comparisons
TOPF	111.25 (10.90)	110.48 (12.02)	110.40 (14.27)	0.05	2, 86	0.951	0.00	–
Digit span	12.05 (3.32)	11.24 (2.86)	10.37 (3.00)	1.99	2, 85	0.144	0.05	–
RCFT-copy	34.27 (1.65)	33.72 (2.15)	33.79 (3.58)	0.56	2, 85	0.572	0.01	–
FAS	45.73 (11.18)	38.64 (11.83)	42.45 (10.04)	3.27	2, 86	0.043	0.07	HW > OB
Animals	25.93 (4.48)	27.60 (8.73)	25.55 (5.08)	0.50	2, 39.71	0.613	0.02	–
TMT (B-A time)	34.15 (18.56)	32.29 (16.41)	36.27 (16.00)	0.16	2, 85	0.849	0.00	–
WCST (pers. errors)	6.23 (4.16)	8.40 (7.01)	7.16 (4.41)	1.09	2, 40.03	0.347	0.03	–
Haying (errors)	0.95 (1.60)	3.56 (4.33)	1.90 (2.17)	5.10	2, 36.42	0.011	0.14	HW > OB

Means are unadjusted (with the exception of TOPF and Digit Span, which represent age-scaled scores) and presented with *SD*

group (inhibition: $p=0.007$; fluency: $p=0.007$). OBED participants committed approximately twice as many inhibition errors, and OB participants nearly four times as many inhibition errors, as HC (only the HC-OB pairwise comparison reached statistical significance, however, $p=0.011$; HC-OBED $p=0.500$). For verbal fluency, OBED participants generated ~ four fewer words, and OB participants ~ seven fewer words in 1 min, than HC (although again, only the HC-OB pairwise comparison reached statistical significance, $p=0.010$; HC-OBED $p=0.299$). No between-group differences were found for the other EF domains (all $ps>0.05$). Analyses were also conducted adjusting for BMI, due to BMI differences between the study groups (Table 4 in "Appendix C"). While EF between-group differences diminished (and did not reach statistical significance with the Sidak correction), the pattern of results remained the same.

Effect of grazing severity

When grazing severity (overall, as well as of the "repetitive eating" and "loss of control" factors independently) was added to analyses for inhibition and phonemic fluency, a differential pattern of results emerged for the three groups (Table 3). Grazing did not influence performance within the HC group (all $ps>0.05$). Within the OB group, however, overall grazing severity as well that of the two subfactors was associated with improved inhibition, such that for every one-point increase in grazing severity, OB participants committed 7–23% fewer errors (GQ total $p<0.001$, GQ "repetitive eating" $p<0.001$, GQ "loss of control" $p=0.023$). No effect of grazing on phonemic fluency was observed in this group. In the OBED group, grazing severity was also associated with better performance; for every point increase in grazing severity, 2–5% more words were generated (GQ total $p=0.031$, GQ "repetitive eating" $p=0.037$, GQ "loss of control" $p=0.010$), with a similar pattern emerging for inhibition, where 9–17% fewer errors were made (although these results were only marginally significant; GQ total $p=0.053$, GQ "repetitive eating" $p=0.040$, GQ "loss of control" $p=0.115$). These analyses were also conducted adjusting for BMI, (Table 5 in "Appendix C"), with results being very similar in terms of direction, strength and statistical significance.

Table 2 Neuropsychological measures adjusted for covariates

Measure	HC ($n=44$) EMM (SE)	OB ($n=25$) EMM (SE)	OBED ($n=20$) EMM (SE)	Wald $\chi^2_{(2)}$	p	Pairwise comparisons
Digit span	11.92 (0.48)	10.73 (0.49)	10.73 (0.68)	3.64	0.162	–
RCFT-copy	34.34 (0.23)	33.57 (0.40)	33.79 (0.78)	3.18	0.204	–
FAS	45.14 (1.37)	38.65 (1.87)	41.20 (1.89)	9.14	0.010	HW > OB
Animals	25.83 (0.63)	27.70 (1.39)	25.00 (1.30)	2.29	0.318	–
TMT(B-A time)	34.75 (2.73)	34.89 (3.23)	32.77 (4.09)	0.18	0.913	–
WCST (pers. errors)	6.34 (0.67)	7.82 (0.97)	6.81 (1.21)	1.57	0.457	–
Haying (errors)	0.91 (0.21)	3.54 (0.91)	1.62 (0.50)	11.00	0.004	HW > OB

Estimated marginal means are adjusted for covariates (age, sex, years of education, depression severity, estimated intellectual functioning)

Table 3 Effect of grazing on phonetic fluency and inhibition performance

Measure	Grazing	HC ($n=44$)		OB ($n=25$)		OBED ($n=20$)	
		IRR [95% CI]	p	*IRR* [95% CI]	p	*IRR* [95% CI]	p
FAS	Total	1.00 [0.99, 1.01]	0.923	1.00 [0.98, 1.02]	0.923	1.02 [1.00, 1.03]	0.031
	Repetitive eating	1.00 [0.98, 1.02]	0.977	1.01 [0.98, 1.04]	0.521	1.02 [1.00, 1.04]	0.037
	Loss of control	1.00 [0.98, 1.02]	0.868	0.99 [0.95, 1.03]	0.567	1.05 [1.01, 1.08]	0.010
Hayling (errors)	Total	0.93 [0.83, 1.05]	0.239	0.87 [0.81, 0.94]	< 0.001	0.91 [0.83, 1.00]	0.053
	Repetitive eating	0.95 [0.83, 1.09]	0.470	0.81 [0.73, 0.90]	< 0.001	0.86 [0.74, 0.99]	0.040
	Loss of control	0.77 [0.57, 1.06]	0.105	0.79 [0.65, 0.97]	0.023	0.83 [0.67, 1.05]	0.115

*IRR*s are adjusted for covariates (age, sex, years of education, depression severity, estimated intellectual functioning)

Discussion

This study aimed to examine EF differences between a group with obesity with and without marked ED psychopathology and a group of HC, and to relate differences to grazing severity within the groups.

The most notable finding was that OB participants displayed substantially lower response inhibition than HC. OBED participants were also less able to inhibit incorrect responses than HC, to a smaller degree. This finding contributes to growing evidence that inhibition is one of the most consistently-affected cognitive domains in obesity [14, 24] while highlighting the need to consider heterogeneity both within obesity and between different facets of inhibition (for example, response inhibition vs impulsive decision-making may be differentially affected in persons with obesity with and without ED [24, 53, 54]). OB participants also had lower phonemic fluency (reflecting frontal lobe activation), but not semantic fluency (reflecting both frontal and temporal activation [55–57]) than HC, with OBED participants placed between the two groups. This finding contributes to an emerging pattern of differences in prefrontal cortex function in obesity. In contrast to Yang et al. [14], no significant differences were found between groups for other EF domains. Our study sample was relatively young and healthy, potentially implicating a lower inflammatory profile and better executive control. Groups were also well-matched on important demographic and cognitive aspects. These factors could also account for the relatively small magnitude difference found between our study groups. However, as obesity has been linked to increased age-related cognitive decline [58], it is possible that differences between the groups could increase over time. The finding of reduced EF in those with obesity has practical implications for the management of high weight, such as that processes other than direct energy intake should be targeted. Treatments that target EF (especially inhibition) such as ImpulsE [40] could be especially effective as a precursor/adjunct to traditional therapies such as behavioural weight loss or cognitive behavioural therapy.

Grazing severity had a modest but statistically significant positive association with phonemic fluency in the OBED group, and with response inhibition in both OB and OBED participants. Previous research has proposed that those with disinhibited eating may exert stronger inhibition at other times as a compensatory mechanism [53]. It is possible that those with obesity and higher EF resources may be able to inhibit impulses to eat large amounts of food and may instead redirect consumption towards smaller amounts of food by through grazing as a strategy to prevent weight gain. The amount consumed through grazing over a long time period may still, however, contribute to weight gain or to the maintenance of high weight. Grazing is also associated with high BMI and poorer mental health [35, 37], therefore this strategy may only be useful in the short term. It is also unclear if the positive association between EF and grazing is more strongly attributable to the "repetitive eating" aspect of grazing or its "loss of control" element, as these factors were highly correlated. It is possible that grazing higher in compulsivity may display a similar pattern of EF associations to binge eating. The relationship between EF and grazing in obesity requires replication within a larger sample size, and further research is needed to clarify directionality. Clinically, it would be useful to assess grazing in persons with high weight and/or ED especially given its high prevalence, and to establish its function, i.e. if this eating pattern contributes to significant overeating, or if it represents a restriction strategy. Given the associations with inhibition found within the current study, it is possible that grazing may serve a restrictive or compensatory purpose. This is important to determine, as it could inform case conceptualisation and treatment approaches, for example whether grazing could be integrated within a treatment model such as CBT-E [59].

This study presents significant strengths, such as adequate selection criteria, well-matched comparison groups, and the use of validated measures. Some limitations were also present; although groups were matched on demographics, the sample was predominantly female; additionally, many of the neuropsychological tests were designed to be used within populations with brain injuries, and may thus not be sensitive at detecting subtler distinctions in EF [60], especially given the relatively small sample size.

Future research could examine moderating factors linking EF and obesity-related behaviours such as grazing, including cognitive load, impulsivity, emotion regulation, and automatic processes such as habit strength, which would better represent "real world" dietary decisions. Secondly, more complex decision-making tasks or tasks incorporating ED-related stimuli could be used. Self-report measures of EF in daily life would also increase ecological validity in terms of EF integration and the complexity of day-to-day functioning [61]. Relationships between EF and grazing should also be examined in clinical ED samples, and using different indices of obesity. Finally, qualitative research would help clarify whether grazing is used as a restrictive strategy deployed to avoid other eating behaviours.

Conclusion

This study found that participants with obesity and without marked ED features manifested lower inhibitory control and phonemic fluency than healthy controls, with the performance of participants with obesity and marked ED features placed between the two groups. In general, grazing severity

was positively associated with better performance for the two groups with obesity, raising the possibility that grazing may be used as a substitutive eating strategy by persons with obesity and with higher EF.

What is already known on this subject?

Poorer executive functioning and a grazing eating pattern have been observed in persons with obesity and in eating disorders. However, the executive functioning correlates of grazing are not known.

What does this study add?

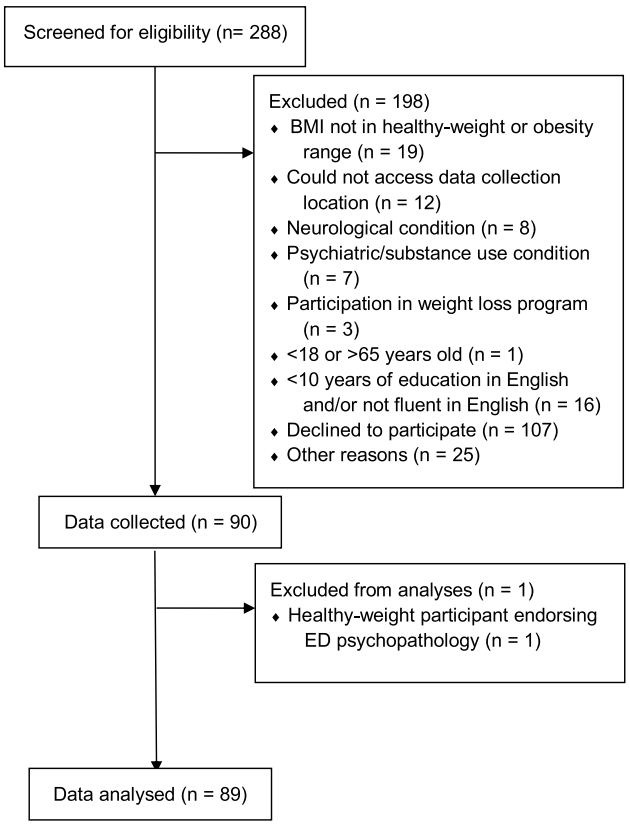
Poorer inhibition and phonemic fluency were found in those with obesity. Grazing had a weak, positive association with these domains in persons with obesity with and without eating disorder features.

Appendix A: Demographic and clinical characteristics

	HC ($n=44$) n%; M(SD)	OB ($n=25$) n%; M(SD)	OBED ($n=20$) n%; M(SD)	F/Welch/ χ^2 statistic	Pairwise comparison
Source of recruitment (community / university)	2 (4.5) / 42 (95.5)	4 (16) / 21 (84.0)	2 (10) / 18 (90)	2.60	–
Age (years)	27.76 (7.45)	30.17 (10.82)	28.44 (8.10)	0.62	–
Sex (female/male/other)	32 (72.7) / 12 (27.3) / 0 (0.0)	13 (52.0) / 12 (48.0) / 0 (0)	14 (70.0) / 6 (30.0) / 0 (0.0)	3.22	–
Ethnicity (Caucasian/Asian/other)	25 (56.8) / 17 (38.6) / 2 (4.5)	14 (56.0) / 10 (40.0) / 1 (4.0)	13 (65.0) / 5 (25.0) / 2 (10.0)	1.95	–
Income (AUD$1000)	61.31 (14.64)	57.29 (10.28)	61.05 (13.61)	0.79	–
Country of birth (Australia/other)	21 (47.7) / 23 (52.3)	13 (52.0) / 12 (48.0)	15 (75.0) / 5 (25.0)	4.26	–
Marital (married or in relationship)	29 (65.9)	9 (36.0)	11 (55.0)	5.76	–
Education (years)	16.78 (2.29)	16.80 (2.76)	15.38 (1.84)	2.82	–
BMI (kg/m^2)	22.31 (2.04)	33.98 (3.11)	38.22 (5.66)	192.75***	OBED > OB > HW
Obesity onset (child/adolescent/ adult)	–	6 (24.0) / 9 (36.0) / 10 (45.5)	2 (10.0) / 6 (30.0) / 12 (60.0)	2.25	–
Alcohol (std. drinks/week)	2.16 (3.18)	2.42 (3.10)	1.81 (3.17)	0.21	–
Smoking (never/past/current)	41 (93.2) / 1 (2.3) / 2 (4.5)	21 (84.0) / 0 (0.0) / 4 (16.0)	15 (75.0) / 1 (5.0) / 4 (20.0)	5.41	–
Cholesterol medication	0 (0.0)	1 (4.0)	0 (0.0)	2.59	–
Blood pressure medication	0 (0.0)	1 (4.0)	0 (0.0)	2.59	–
Antidepressant medication	1 (2.3)	1 (4.0)	3 (15.0)	4.37	–
Trying to lose weight	4 (9.1)	19 (76.0)	16 (80.0)	42.71***	OB/OBED > HW
Physical activity (< 1 h/1–5 h/ > 5 h per week)	6 (13.6) / 25 (56.8) / 13 (29.5)	5 (20.0) / 17 (68.0) / 3 (12.0)	3 (15.0) / 15 (75.0) / 2 (10.0)	4.97	–
EDE-Q Global Score	0.67 (0.65)	1.75 (0.82)	3.41 (0.70)	102.13***	OBED > OB > HW
Objective binge episodes (no.)	0.70 (3.11)	1.36 (2.45)	14.45 (20.93)	13.73***	OBED > HW/OB
Objective binge episodes (presence)	5 (11.4)	9 (36.0)	20 (100.0)	45.83**	OBED > OB > HW
Compensatory behaviour presence	7 (15.9)	9 (36.0)	9 (45.0)	6.84*	OBED > HW
Depression (DASS-21)	2.05 (3.55)	1.96 (2.13)	7.30 (5.42)	8.78**	OBED > HW/OB
Grazing (GQ)	7.23 (4.61)	11.52 (4.98)	16.10 (5.57)	23.02***	OBED > OB > HW
Grazing- repetitive eating	5.11 (3.23)	7.04 (3.26)	8.80 (3.40)	9.21***	OB/OBED > HW
Grazing-loss of control	2.11 (2.10)	4.48 (2.29)	7.30 (2.45)	37.91***	OBED > OB > HW

* $p<0.05$, ** $p<0.01$, *** $p<0.001$

Appendix B: Flow diagram



Appendix C: Analyses adjusted for covariates and participant BMI

See Tables 4, 5

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Author contributions AIH and ST designed the study. AIH conducted literature searches, data collection, statistical analysis and drafted the manuscript. PH provided input into the statistical analysis and analysis interpretations. All authors contributed to and have approved the final manuscript.Credit author statement AIH: Conceptualization; Methodology; Formal analysis; Investigation; Writing–Original Draft; Project administration; Funding acquisition. PH: Methodology; Resources; Writing–Review and Editing. ST: Conceptualization; Methodology; Resources; Writing–Review and Editing; Funding acquisition.

Table 4 Neuropsychological measures adjusted for covariates and BMI

Measure	HC ($n=44$) EMM(SE)	OB ($n=25$) EMM(SE)	OBED ($n=20$) EMM(SE)	Wald $\chi^2_{(2)}$	p	Pairwise comparisons
Digit span	11.90 (0.73)	10.75 (0.62)	10.75 (0.91)	0.99	0.611	–
RCFT-copy	32.95 (0.99)	34.73 (0.97)	35.53 (0.87)	3.43	0.180	–
FAS	42.38 (2.20)	40.48 (2.13)	44.59 (3.27)	1.99	0.370	–
Animals	25.71 (1.26)	27.79 (1.47)	25.15 (1.86)	2.43	0.297	–
TMT(B-A time)	42.07 (4.69)	28.81 (4.24)	23.82 (4.91)	4.47	0.107	–
WCST (pers. errors)	7.37 (1.44)	6.91 (1.31)	5.62 (1.29)	1.11	0.573	–
Haying (errors)	1.16 (0.35)	2.83 (0.94)	1.22 (0.54)	3.21	0.201	–

Estimated marginal means are adjusted for covariates (age, sex, years of education, depression severity, estimated intellectual functioning) and BMI

Table 5 Effect of grazing on phonetic fluency and inhibition performance adjusted for covariates and BMI

Measure	Grazing	HC ($n=44$)		OB ($n=25$)		OBED ($n=20$)	
		IRR [95% CI]	*p*	*IRR* [95% CI]	*p*	*IRR* [95% CI]	*p*
FAS	Total	1.00 [0.99, 1.01]	0.723	1.00 [0.98, 1.02]	0.883	1.01 [1.00, 1.03]	0.029
	Repetitive eating	1.00 [0.98, 1.02]	0.894	1.01 [0.98, 1.05]	0.458	1.02 [1.00, 1.04]	0.080
	Loss of control	1.01 [0.98, 1.03]	0.576	0.99 [0.95, 1.03]	0.553	1.04 [1.01, 1.07]	0.009
Hayling (errors)	Total	0.92 [0.78, 1.08]	0.319	0.90 [0.84, 0.96]	0.001	0.91 [0.79, 1.05]	0.188
	Repetitive eating	0.95 [0.82, 1.09]	0.458	0.82 [0.73, 0.91]	< 0.001	0.86 [0.73, 1.03]	0.095
	Loss of control	0.77 [0.57, 1.05]	0.101	0.78 [0.65, 0.94]	0.007	0.84 [0.65, 1.08]	0.172

*IRR*s are adjusted for covariates (age, sex, years of education, depression severity, estimated intellectual functioning) and BMI

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Data availability The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Code availability N/A.

Compliance with ethical standards

Conflict of interest AIH has no competing interests to declare. PH has received in sessional fees and lecture fees from the Australian Medical Council, Therapeutic Guidelines publication, and New South Wales Institute of Psychiatry and royalties from Hogrefe and Huber, McGraw Hill Education, and Blackwell Scientific Publications, and she has received research grants from the NHMRC and ARC. She is Chair of the National Eating Disorders Collaboration in Australia (2012–2013). In July 2017 she provided a commissioned report for Shire Pharmaceuticals on lisdexamfetamine and binge eating disorder and in 2018 received honoraria for education of Psychiatrists. ST has received royalties from Hogrefe and Huber, McGraw Hill Education and Routledge for the publication of books/chapters. He is the Chair of the Shire (Australian) BED Advisory Committee and has received travel grants, research grants and honoraria from Shire for commissioned reports and is a consultant to Weight Watchers International.

Ethical approval This study was performed in line with the principles of the Declaration of Helsinki. The study was approved by the University of Sydney Human Research Ethics Committee (Approval no.: 2014/936).

Informed consent Written consent to participate was obtained from all study participants.

Consent for Publication All participants have consented to having deidentified, group data published in a journal article.

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