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# **Temporal discounting and the tendency to delay gratification across the eating disorder spectrum**

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## **Abstract**

Bulimia nervosa (BN) and binge eating disorder (BED) have been associated with poorer reward-related inhibitory control, reflected by a reduced tendency to delay gratification. The opposite has been reported in anorexia nervosa (AN), but differences have not been directly compared across eating disorders (EDs). This study investigated self-reported (delaying gratification inventory) and task-based (temporal discounting) inhibitory control in 66 women with an ED and 28 healthy controls (HC). Poorer task-based inhibitory control was observed in the BN compared to the AN group, and poorer self-reported inhibitory control in the BN and BED groups compared to the AN and HC groups, suggesting that reward-related inhibitory control varies across EDs. Symptom severity correlated with poorer self-reported (but not task-based) inhibitory control across the EDs. These data provide some support for transdiagnostic mechanisms, and highlight the importance of addressing perceived loss of control in the treatment of EDs.

## **Introduction**

The aetiology of eating disorders (EDs) is complex, and the mechanisms underlying their development and maintenance are unclear (Kaye et al., 2015, Wu, Hartmann, Skunde, Herzog & Friederich, 2013). However, many hypotheses implicate alterations in behavioural control and reward processing as key factors. For example, many of the current models have dysfunctional fronto-striatal neural circuits involved in reward and self-regulatory processing as central elements (Berner & Marsh, 2014, Marsh, 2009, O'Hara, Campbell & Schmidt, 2015), circuits that are also thought to be involved in intertemporal decision-making (Peters & Büchel, 2011). Part of the evidence supporting dysfunctional behavioural control and reward processing in the development and maintenance of EDs comes from reports of altered performance on neuropsychological questionnaire and task-based measures of inhibitory control in individuals with EDs (for reviews, see Bartholdy, Dalton, O'Daly, Campbell & Schmidt, 2016, Fischer, Smith & Cyders, 2008, Guillaume et al., 2015, McClelland et al., 2016, Schag, Schönleber, Teufel, Zipfel & Giel, 2013a, Waxman, 2009, Wierenga et al., 2014, Wu et al., 2013). However, the complexity, and to some extent the validity, of such models is further complicated as there is a diagnostic and aetiological issue related to the extent to which the EDs can be seen as discrete illnesses, or whether they are better suited to being conceptualised within a transdiagnostic framework. In the present context where issues related to self-regulation are being considered, it is of note that models have emerged characterising EDs along a spectrum, with AN and BED thought to have the highest and lowest levels of behavioural control, respectively (Brooks, Rask-Andersen, Benedict & Schiöth, 2012).

Reward-related inhibitory control has been assessed using temporal discounting (TD) paradigms that investigate the degree to which a reward is devalued (discounted) over time. In such tasks, TD is reflected by a preference for smaller rewards received sooner than larger delayed rewards, and is proposed to reflect choice impulsivity and poor reward-related inhibitory control (Bari & Robbins, 2013). Studies in EDs have reported that compared to healthy adults, those with bulimia nervosa (BN; Kekic et al., 2016) and binge eating disorder (BED; Davis, Patte, Curtis & Reid, 2010, Manasse

et al., 2015a, Manasse et al., 2015b, Manwaring, Green, Myerson, Strube & Wilfley, 2011, Mole et al., 2015) show greater TD (i.e., a preference for smaller-sooner rewards), whereas the opposite has been reported in studies of adults with anorexia nervosa (AN; Decker, Figner & Steinglass, 2015, Steinglass et al., 2012). These findings are consistent with core symptoms of these disorders. For example, binge eating can be regarded as a manifestation of the tendency to act in pursuit of immediate pleasure-driven desires, as patients with BN and BED appear to have heightened reward sensitivity to food cues (Brooks et al., 2011, Schag et al., 2013b). Thus, the presence of either relatively high or low levels of TD may be a risk factor or marker for the development and maintenance of EDs, and as such, could be a target for behavioural interventions. Further research exploring the specificity of TD alterations across ED symptoms and diagnoses will provide insight into possible transdiagnostic mechanisms of TD. To date, however, there have been no direct comparisons in TD behaviour across the different EDs.

This study is the first to compare the tendency to delay gratification in individuals across the ED spectrum. Following on from our previous study comparing individuals with BN with healthy controls (Kekic et al., 2016), the tendency to delay gratification was assessed using both a questionnaire assessment and a neuropsychological task paradigm. It was hypothesised that individuals with AN would have the greatest tendency to delay gratification both in task-based measures (i.e., reduced TD [preference for larger-later rewards]) and in self-reports compared to the BN and BED groups and healthy controls. Secondly, it was hypothesised that the BN and BED groups would be similar in their tendency to delay gratification, and that both groups would show a poorer tendency to delay gratification (i.e., greater TD) compared to healthy controls.

## **Materials and methods:**

### **Participants:**

A total of 66 women meeting DSM-5 criteria for an ED (28 AN [12 restrictive subtype; ANR, 16 binge-purge subtype; ANBP], 27 BN, 11 BED,) and 28 healthy non-dieting women (healthy controls; HC) participated in the study. Healthy participants were recruited through online and poster advertisements at King's College London. Participants with an ED were recruited via online and poster advertisements at King's College London and the Eating Disorder Unit at the South London and Maudsley NHS Foundation Trust, as well as via online adverts on the Beat website (the UK's national eating disorder charity), and a research recruitment website ([www.callforparticipants.com](http://www.callforparticipants.com)). ED diagnoses were confirmed using the Eating Disorder Diagnostic Scale (EDDS; Stice, Telch & Rizvi, 2000) and the screening module of the Structured Clinical Interview for DSM-IV Disorders, Researcher Version (SCID-IV; First, Spitzer, Gibbon & Williams, 2002). Participants in the ED groups were required to meet one of the following criteria: (a) DSM-5 diagnosis of AN and a body mass index (BMI) of  $\leq 19\text{kg/m}^2$ , (b) DSM-5 diagnosis of BN and a BMI of  $>18.5\text{kg/m}^2$ , or (c) DSM-5 diagnosis of BED and a BMI of  $>18.5\text{kg/m}^2$ . Participants taking psychotropic medication were included provided their medication had been stable for at least 14 days prior to the study. Healthy controls were required to be of healthy BMI ( $20\text{-}25\text{kg/m}^2$ ) and have never had an eating disorder or any current/previous neurological or major psychiatric disorder. Participants were excluded if they were using illicit drugs or were pregnant.

### **Procedure:**

Participants were assessed for eligibility using a telephone screening assessment. This involved confirmation of ED diagnosis (EDDS, SCID-IV) and other inclusion/exclusion criteria (using a non-validated study-specific screening questionnaire). Eligible participants were invited to come for a single research visit to the Institute of Psychiatry, Psychology and Neuroscience at King's College London. During this visit, they completed a questionnaire battery including the Eating Disorder

Examination Questionnaire (EDE-Q; Fairburn, 2008, Fairburn & Beglin, 1994) to assess clinical characteristics, the Depression, Anxiety and Stress Scale (DASS-21; Lovibond & Lovibond, 1995) to assess mood and the Delaying Gratification Inventory (DGI; Hoerger, Quirk & Weed, 2011) to assess self-reported tendency to delay gratification, and a 10cm visual analogue scale to assess current hunger (“How hungry are you at the moment?”, with answers ranging from “not at all hungry” to “extremely hungry”). Following this, participants completed a hypothetical monetary TD task (Rubia, Halari, Christakou & Taylor, 2009) during which they indicated their preference between a variable smaller immediate reward (£0-£100) and a larger delayed reward (fixed at £100) over 100 binary choices (25 choices at each of 4 delays: 1 week, 1 month, 1 year, 2 years). Finally, height and weight were measured. Participants were debriefed and compensated with up to £15 for their time and travel.

### **Statistical analysis:**

TD was quantified by plotting the mean indifference point for each subject (i.e., the point at which both choices are valued equally) against time delay and summing the area of the trapezoids under the curve (area under the curve; AUC). The area of each trapezoid was calculated using the following equation:  $(x_2 - x_1) / ([y_1 + y_2] / 2)$ , where  $x_2$  and  $x_1$  are the successive delays (expressed as a proportion of the maximum delay (e.g., 1 week = 1/104, 1 month = 1/24, 1 year = 1/12, 2 years = 1/6)), and  $y_1$  and  $y_2$  are the respective standardised subjective values (i.e., the indifference points expressed as a proportion of the maximum available reward value) (Myerson, Green & Warusawitharana, 2001).

Three one-way ANOVAs were calculated to explore the effect of group (AN, BN, BED, HC) on BMI, TD (AUC) and on DGI total and money subscale scores. Bonferroni-corrected post-hoc t-tests were conducted to explore group differences. Kruskal-Wallis one-way ANOVAs were employed to assess group differences in age, ED pathology (EDE-Q Global score) and general psychopathology, as these data were non-normally distributed. Spearman and Pearson correlations were conducted to explore the relationship between potential confounding factors (BMI, age, level of education, length of illness, general psychopathology [DASS Total scores]) and the tendency to delay gratification (TD [AUC] and DGI [Total and Money subscale scores]). Correlated variables were included as nuisance

covariates into the General Linear Models to explore confounding effects. Purge frequency was the only measure of ED symptom severity that was normally distributed. Pearson and Spearman's correlations were also conducted to assess the association between ED symptom severity (EDE-Q Global score; frequency of binge eating; frequency of purging [sum of reported frequency of self-induced vomiting, laxative use and diuretic use]; lowest BMI at current height) and the tendency to delay gratification across the 3 ED groups.

**Ethical Approval:**

This study was approved by London-Bloomsbury Research Ethics Committee (REC Ref: 15/LO/0251). Written informed consent was obtained from all participants.



## **Results:**

### **Demographic and clinical information**

Participant demographic and clinical characteristics are presented in Table 1. The 4 groups did not differ according to age ( $\chi^2(3)=4.590, p=0.204$ ) or hunger ( $\chi^2(3)=6.065, p<0.109$ ). Unsurprisingly, significant group differences were observed for BMI,  $F(3,90)=37.081, p<0.001$ . Bonferroni-corrected post-hoc t-tests revealed this was due to significantly lower mean BMI in the AN group compared to the HC ( $t(54)=-9.047, p<0.001$ ), BN ( $t(53)=-7.550, p<0.001$ ) and BED ( $t(10.887)=-5.667, p<0.001$ ) groups, and to significantly higher BMI in the BED group compared to the HC group ( $t(10.681)=-3.218, p=0.008$ ).

Group differences in global ED pathology (EDE-Q Global score), general psychopathology (DASS Total Score) and length of illness were assessed across the 3 ED groups. These analyses revealed no differences between the groups on global ED pathology ( $\chi^2(2)=3.437, p=0.179$ ) or general psychopathology ( $\chi^2(2)=0.348, p=.840$ ), however did reveal significant group differences for length of illness ( $\chi^2(2)=14.163, p=0.001$ ). Bonferroni-corrected post-hoc Mann-Whitney U Tests revealed that this was driven by a significantly longer duration of illness in the AN group compared to the BN ( $U=215.5, z=-2.745, p=0.006$ ) and BED ( $U=51.0, z=-3.231, p=0.001$ ) groups, and a trend towards a shorter illness duration in the BED group compared to the BN group ( $U=95.5, z=-1.724, p=0.085$ ). Of those who reported binge eating, frequency of binge episodes did not differ between diagnostic groups ( $\chi^2(2)=2.466, p=0.291$ ). Similarly, purge frequency did not significantly differ between individuals with ANBP and individuals with BN ( $t(11.494)=1.724, p=0.111$ ).

A large proportion (48.5%) of individuals in the ED groups reported they were taking medications at the time of the scan. Of the 19 participants with AN who reported taking medication, 12 were on a stable dose of one or more psychotropic medications (11 were taking antidepressants), while the remaining 7 reported taking vitamin supplements (including iron or calcium tablets), antibiotics for

dermatological reasons, and sleeping tablets. In the BN group, 8 participants reported they were taking medication: 5 were on a stable dose of psychotropic medication(s) including antidepressant medication, 1 reported taking painkillers for acid pain, 1 reported taking beta-blockers and steroids, and 1 did not specify their medication. Five participants in the BED group reported taking medications, although only 1 was receiving psychotropic medication (antidepressants): the remaining 4 disclosed medications for high blood pressure, high cholesterol, and physical problems such as joint mobility (in the knee) and bladder control.

Table 1. Demographic and clinical information

	HC (n=28)	AN (n=28)	BN (n=27)	BED (n=11)
<b>Demographic information</b> (mean [SD])				
Age (years)	24.64 (5.14)	30.00 (10.51)	25.30 (6.85)	28.73 (11.33)
BMI (kg/m <sup>2</sup> )	22.04 (2.03)	16.79 (2.31)	22.57 (3.30)	28.86 (6.92)
Right handed (%)	82.14%	78.57%	96.30%	81.81%
% completed undergraduate degree	100.00%	77.78%	79.17%	88.89%
<b>ED-related variables</b> (mean [SD])				
EDE-Q Global	0.53 (0.66)	3.80 (1.29)	4.29 (1.00)	3.65 (1.08)
Self-reported length of illness (years)	-	10.59 (10.04)	5 (5.51)	2.32 (3.99)
Lowest BMI at current height (kg/m <sup>2</sup> )	-	13.76 (2.32)	18.44 (2.70)	22.00 (4.64)
Binge eating episodes (n reported in preceding 28 days [mean frequency [SD]])	0 (0.00 [0.000])	8 (11.14 [22.34])	27 (18.04 [22.78])	11 (8.00 [5.15])
Self-induced vomiting episodes (n reported in preceding 28 days [mean frequency [SD]])	0 (0.00 [0.000])	9 (28.19 [82.78])	19 (16.94 [20.48])	0 (0.00 [0.00])
Laxative use (n reported in preceding 28 days [mean frequency [SD]])	0 (0.00 [0.000])	5 (9.07 [28.20])	12 (6.00 [9.90])	0 (0.00 [0.00])
Diuretic use (n reported in preceding 28 days [mean frequency [SD]])	0 (0.00 [0.000])	0 (0.00 [0.00])	4 (1.70 [5.60])	0 (0.00 [0.00])
n reporting comorbid diagnoses (%)	0 (0%)	15 (53.2%)	9 (33.3%)	2 (18.2%)
n currently taking medication <sup>a</sup> (% , n taking antidepressants)	0 (0%)	19 (67.9%, 11)	8 (29.6%, 5)	5 (45.5%, 1)
<b>Hunger</b> (mean [SD])				
Visual analogue scale	0.31 (0.25)	0.20 (0.23)	0.28 (0.28)	0.40 (0.31)
<b>General psychopathology</b> (mean [SD])				
DASS-Total	5.89 (5.59)	28.18 (15.09)	29.41 (15.84)	26.82 (16.58)

<sup>a</sup>Not including oral or internal contraceptive medication.

### Self-reported tendency to delay gratification

The data from self-report (DGI) and task-based assessments of the tendency to delay gratification are reported in table 2. A main effect of group was observed for self-reported reward-related impulsivity (DGI total:  $F(3,90)=7.475$ ,  $p<0.001$ ; DGI money subscale:  $F(3, 90)=2.982$ ,  $p=0.036$ ). Bonferroni-corrected post-hoc t-tests showed that both the BN and the BED groups demonstrated a poorer overall tendency to delay gratification (DGI total) compared to HC ( $t(53)=3.345$ ,  $p=0.002$ , and  $t(37)=3.725$ ,  $p=0.001$ , respectively) and to women with AN ( $t(53)=3.201$ ,  $p=0.002$ , and  $t(35)=3.377$ ,  $p=0.002$ , respectively). Participants with BN also showed a poorer tendency to delay gratification specifically with respect to money (DGI Money subscale scores) compared to AN ( $t(53)=2.560$ ,  $p=0.013$ ) and HC (trend:  $t(53)=1.749$ ,  $p=0.086$ ), however these comparisons did not survive Bonferroni correction.

Table 2. Neuropsychological data

	HC (n=28)	AN (n=28)	BN (n=27)	BED (n=11)
<b>Delaying gratification</b> (mean [SD])				
DGI Total	132.54 (13.29)	133.46 (17.33)	117.69 (19.21)	113.73 (16.38)
DGI Money	25.29 (3.86)	26.36 (4.09)	22.83 (5.27)	23.45 (6.42)
<b>Temporal Discounting (TD)</b> (mean [SD])				
AUC of trapezoids	0.63 (0.22)	0.78 (0.20)	0.59 (0.22)	0.64 (0.21)

### Temporal discounting task

A main effect of group was observed for TD performance ( $F(3,90)=3.838$ ,  $p=0.012$ ). Bonferroni-corrected post-hoc t-tests revealed that this effect was driven by greater TD in BN women compared to women with AN,  $t(53)=3.217$ ,  $p=0.002$ . No other group differences in TD behaviour were observed.

### Potential confounding variables

As can be seen in Table 3, general psychopathology (DASS Total score), but not age or level of education, correlated with DGI Total scores ( $p<0.001$ ). BMI showed a trend towards a correlation with DGI Total ( $p=0.079$ ) and DGI Money subscale scores ( $p=0.078$ ). None of these variables

correlated with TD performance (AUC) (all  $p > 0.1$ ). Including general psychopathology nuisance covariate did not affect the DGI total findings ( $F(4,89)=7.967$ ,  $p < 0.001$ , and nor did BMI affect findings regarding DGI Total ( $F(4,89)=3.269$ ,  $p=0.015$ ), however group differences in DGI Money subscale scores were no longer significant ( $F(4,89)=1.941$ ,  $p=0.110$ ).

Table 3. Correlation coefficients between demographic and clinical information and neuropsychological outcomes

		1	2	3	4	5	6	7
Age	1							
Level of education	2	.225*	-					
Length of illness	3	.260*	-.246*	-				
DASS-21 Total	4	.053	-.537**	.591**	-			
BMI	5	-.036	.051	-.219*	-.048	-		
DGI Total	6	.056	.158	-.160	-.396**	<b>-.182</b>	-	
DGI Money	7	.022	.018	-.073	-.154	<b>-.183</b>	.569**	-
AUC	8	.140	.073	.114	.037	-.170	.241*	.218*

\* $p < 0.05$

\*\* $p < 0.01$

**Bold** =  $p < 0.1$

### Correlations with ED symptom severity

Amongst the ED groups, 48 individuals reported binge eating at least once in the preceding 28 days (13 AN, 24 BN, 11 BED) and 35 reported purging (12 AN, 23 BN, 0 BED). Somewhat surprisingly, 3 individuals in the BN group did not report binge eating or purging in the month preceding the study session although they did meet criteria for BN at the time of screening, which assessed the average number of binge eating and purging episodes within the previous 3 months. One additional individual in the BN group reported binge eating but not purging in the month prior to the study session.

Across the ED groups, binge frequency negatively correlated with self-reported tendency to delay gratification (DGI Total:  $r_s = -.307$ ,  $p=0.030$ ; DGI Money:  $r_s = -.287$ ,  $p=0.044$ ) but not with task-based TD performance (AUC:  $r_s = -.169$ ,  $p=0.242$ ). This suggests that individuals who report a greater frequency of binge eating episodes also report a reduced tendency to delay gratification, but do not show more impulsive money choice behaviour. Purge frequency did not correlate with any measure of the tendency to delay gratification (DGI Total scores:  $r = .020$ ,  $p=0.907$ ; DGI Money subscale scores:

$r = -.093, p = 0.583$ ; AUC:  $r = -.017, p = 0.921$ ). EDE-Q Global scores were significantly negatively correlated with DGI Total ( $r_s = -.373, p = 0.002$ ) and DGI Money subscale scores ( $r_s = -.428, p < 0.001$ ), but not with TD performance ( $r_s = -.158, p = 0.204$ ). In contrast, lowest BMI at current height negatively correlated with TD performance ( $r_s = -.294, p = 0.019$ ), but not with DGI Total ( $r_s = -.203, p = 0.112$ ) or Money subscale scores ( $r_s = -.084, p = .511$ ).

## **Discussion:**

This study is the first to compare the tendency to delay gratification in women who have diagnoses across the ED spectrum. Our two hypotheses were a) that individuals with AN would show the greatest tendency to delay gratification both on task-based measures (i.e., reduced TD [preference for larger-later rewards]) and in self-reports (DGI scores) compared to the HC, BN and BED groups, and b) that both the BN and BED groups would be similar in their tendency to delay gratification, and that these groups would show a poorer tendency to delay gratification (i.e., greater TD) compared to HC. These hypotheses were substantially supported in that self-reported impulsivity (i.e., lower DGI scores) was greater in women with BN and BED compared with AN, although higher rates of TD (i.e., lower AUC values) were only observed in the women with BN. The data suggest that in the context of reward, the subjective perception of impulsivity or loss of control, rather than conscious decision making, is important in BED. However, this may also simply be a consequence of the small sample size of the BED group in this study, and therefore replications are required to determine the reliability of these findings.

The present findings are consistent with previous reports of altered reward-related behavioural control in EDs and provide some support for spectrum models (Brooks et al., 2012), with greater reward-related impulsivity being present in women with BN and BED compared to the exaggerated tendency to delay gratification observed in women with AN. It is of note that the AN group, by chance, had a longer average duration of illness than the other groups, which may have contributed to differences between groups, although length of illness was not associated with self-report or task-based assessment of TD. Although the AN group in the present study was comprised of participants with either the restrictive or binge-purge subtype, the sample size for each diagnostic subtype was limited, and did not offer sufficient power to detect subtype-specific effects. Previous studies comparing the AN subtypes (ANR vs. ANBP) revealed that only individuals with the restrictive subtype showed enhanced TD compared to HC, but no differences were observed between ANBP and either ANR or HC (Decker et al., 2015, Steinglass et al., 2012). Thus, altered TD may not be the most sensitive

marker of general eating-related pathology (i.e., distinguishing individuals with an ED from those without), however it may constitute a useful marker to distinguish between the different EDs. Future studies are required to explore differences between the diagnostic subtypes on temporal discounting capacity and the self-reported tendency to delay gratification.

While the results were in the direction predicted on the basis of previous research (e.g., Decker et al., 2015, Kekic et al., 2016, Manwaring et al., 2011, Steinglass et al., 2012), none of the ED groups showed differences in TD or self-reported tendency to delay gratification compared to HC. It is possible that the number of participants within each of the groups in the present study may have been insufficient to detect differences, although it is of note that null findings have been reported (e.g., Manasse et al., 2014, Ritschel et al., 2015). For example, Davis et al. (2010) reported that group differences in TD between obese individuals with and without BED compared to HC were no longer apparent after controlling for education.

Global ED pathology, binge frequency and BMI were inversely associated with DGI total scores, but not with TD performance, whereas greater general psychopathology (assessed by a combined measure of depression, anxiety and stress) was associated with poorer outcomes on both the task-based and self-report measures. This suggests that participants reporting more severe symptoms at the time of the assessment perceive themselves to have a poorer overall tendency to delay gratification, although this is not reflected in their behavioural performance. This is similar to the findings in individuals with BED, whose self-reports were discrepant from task-based assessments. Nonetheless, given that these correlations emerged from a trans-diagnostic assessment of global pathology and the frequency of symptoms that can be present across all three EDs, the perception or experience of a loss of control (regardless of observable behavioural performance) may be a shared vulnerability factor across the EDs.

While loss of control is reflected clinically in some of the core symptoms of EDs such as binge eating, it may be that the psychological *perception* of loss of control is an important and distinct factor from

the observable behavioural counterpart. For instance, the experience of a loss of control can promote feelings of guilt and negative affect, which may trigger binge-eating episodes as a method of coping (Berg et al., 2015). Together, the findings from the present study suggest the need for psychological treatments to directly address the patient's self-perceptions of their tendency to control their behaviour. However it is not clear whether poorer perceived self-control is a consequence of the severity of the disorder or is a precursor that could be used to identify individuals who may subsequently develop a more severe course of illness.

## **Conclusion**

Our data show that the tendency to delay gratification (one aspect of reward-related inhibitory control), and thus the mechanisms underlying reward-related inhibitory control, is not uniform across the EDs: individuals with BN and BED report a reduced ability to delay gratification compared to AN and HC. However, no difference was observed between women with AN and HC, and behavioural differences in temporal discounting were only observed between the AN and BN groups, therefore the effect may be subtle. Moreover, the discrepancy in the tendency to delay gratification between self-report and task-based measures in individuals with more chronic EDs or with BED suggests the importance of the perception of impulsivity/loss of control. This, and the observed correlations between transdiagnostic ED symptoms, ED chronicity and the tendency to delay gratification suggest that shared mechanisms exist. Linear spectrum models of EDs are useful in the development of hypotheses, and may also be useful clinically. The present findings provide some support for spectrum models, but suggest that they are unlikely to be universally applicable with respect to the multifaceted nature of inhibitory control alterations that have been reported in the various EDs. Indeed, systematic reviews exploring specific aspects of inhibitory control across the EDs are emerging (e.g., Bartholdy et al., 2016, McClelland et al., 2016), which describe how different elements of inhibitory control may contribute to various disordered eating behaviours and how these may be differentially altered across the diagnostic categories. The present study highlights the need for direct comparisons of inhibitory control abilities and experiences across the different EDs to



improve understanding of the mechanisms and appreciate the complexity through which inhibitory control may contribute to the aetiopathology of EDs.

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