

## Original Research

**Cite this article:** Boswell RG, and Grilo CM (2021). General impulsivity in binge-eating disorder. *CNS Spectrums* 26(5), 538–544. <https://doi.org/10.1017/S1092852920001674>

Received: 24 March 2020

Accepted: 09 July 2020

## Key words

Binge-eating disorder; impulsivity; comorbidity; obesity; eating disorders.

## Author for correspondence:

Rebecca G. Boswell, PhD

Email: [rebecca.boswell@yale.edu](mailto:rebecca.boswell@yale.edu)

## Abstract

**Background.** The nature and significance of impulse-control difficulties in binge-eating disorder (BED) are uncertain. Most emerging research has focused on food-specific rather than general impulsivity. The current study examines the clinical presentation of patients with BED categorized with and without clinical levels of general impulsivity.

**Method.** A total of 343 consecutive treatment-seeking patients with BED were categorized as having BED with general impulsivity (GI+; N = 73) or BED without general impulsivity (GI–; N = 270) based on structured diagnostic and clinical interviews. The groups were compared on demographic, developmental, and psychological features, and on rates of psychiatric and personality comorbidity.

**Results.** Individuals with BED and general impulsivity (GI+) reported greater severity of eating-disorder psychopathology, greater depressive symptoms, and greater rates of comorbidity than those without general impulsivity (GI–).

**Conclusions.** A subtype of individuals with BED and general impulsivity may signal a more severe presentation of BED characterized by heightened and broader psychopathology. Future work should investigate whether these impulse-control difficulties relate to treatment outcomes.

## Introduction

Binge-eating disorder (BED) is a relatively common eating disorder (most recent prevalence estimate of roughly 1% among adults in the United States<sup>1</sup>) that is associated with psychosocial impairment.<sup>2</sup> BED is characterized by a recurrent pattern of binge eating (i.e., consuming large quantities of food in a short period of time while experiencing a subjective sense of loss of control) associated with marked distress and without weight-compensatory behaviors.<sup>3,4</sup> One factor thought to contribute to the development and maintenance of binge-eating psychopathology is impulsivity, a multidimensional construct that reflects a propensity to engage in increased reward-seeking behavior and poor reward-related decision-making.<sup>5</sup> Impulsivity differs from compulsivity,<sup>6</sup> although obsessions/compulsions about food are associated with frequency of binge eating and BED severity.<sup>7</sup> Notably, impulsivity has been associated with increased risk of developing eating disorders<sup>8,9</sup> and with poorer outcomes in a small pilot intervention for BED.<sup>10</sup>

Research has found that individuals with BED have poor impulse control, specifically in the context of food stimuli. Compared to groups with overweight, individuals with BED show greater food-reward sensitivity and greater rash-spontaneous behavior in the context of food.<sup>11–13</sup> Experimental, neurocognitive, and neuroimaging studies have found that impulsivity-related deficits in individuals with BED may be food-specific.<sup>14–17</sup> Accordingly, researchers have begun to develop and test interventions for BED that target food-related impulsivity; to date, however, these initial efforts have yielded mixed results.<sup>18,19</sup>

In contrast, a substantial body of research suggests that individuals with BED show impulsivity across multiple contexts. Individuals with eating psychopathology engage in a range of impulsive behaviors, including substance misuse,<sup>20</sup> nonsuicidal self-injury,<sup>21</sup> shoplifting,<sup>22</sup> and risky sexual behavior.<sup>23</sup> Both experimental and neuroimaging studies have found evidence of general reward sensitivity and rash impulsiveness outside of food-related context in BED.<sup>16,24–27</sup> Consistent with this, individuals with BED report elevated scores on trait measures of impulsivity.<sup>28</sup> Further, BED frequently co-occurs with impulsivity-related psychopathology, including mood, substance, and personality disorders.<sup>29–35</sup> More specifically, analysis of the National Comorbidity Survey Replication data revealed that 43.3% of persons with BED reported comorbid impulse control disorders.<sup>31</sup> Because there is a shared neurobiological basis of many impulsive behaviors,<sup>13,36–38</sup> a general impairment in impulse control could underlie both BED and related comorbidities. If so, general impulse control could represent an important broader target to include in or to refine treatments for BED.

Subtyping individuals with BED based on patterns of impulsivity might inform treatment formulations and intervention efforts. Previous work subtyping BED has focused on the severity of clinical presentation for groups differentiated by dietary restraint and negative affect<sup>39</sup> or differentiated by rates of psychiatric comorbidity.<sup>40</sup> An older body of research identified a

subgroup of individuals with bulimia nervosa (BN) characterized with “multi-impulsivity”<sup>41</sup> who exhibited a more severe clinical presentation characterized by greater distress, more severe cognitive eating-related psychopathology, greater rates of comorbidity, and a poorer response to treatment.<sup>42-45</sup> These “multi-impulsive” individuals seemed susceptible to “behavioral substitution” during eating-disorder treatment, such that reductions in binge eating and purging were associated with increases in other impulsive behaviors (eg, substance use).<sup>46</sup>

Given that individuals with BED have shown impulse control difficulties in various contexts,<sup>16,24-27</sup> it is possible that a “multi-impulsive” subtype may represent a more severe variant of BED. We are unaware of prior research on the frequency and clinical presentation of an impulsivity-related subtype of BED. Thus, we compared treatment-seeking patients with BED categorized with and without impulsivity in more than two domains (general impulsivity) in eating-related psychopathology and in rates of psychiatric comorbidity.

## Methods

### Subjects

Participants in this report were a consecutive series of 343 treatment-seeking patients at one site (Yale University School of Medicine) evaluated between 1996 and 2001 who met Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV)<sup>3</sup> research criteria for BED. Previous publications with this dataset have characterized psychiatric and personality disorder comorbidity<sup>30,47</sup>; the data presented here, which focuses on subtyping by impulsivity, is entirely distinct from the previous reports. The majority of participants were female (74.6%), white (80.7%), married (66.9%), and had completed at least some college-level education (84.4%). Participants ranged in age from 18 to 60 ( $M = 44.70$ , standard deviation [ $SD$ ] = 9.26; previously reported<sup>30,47</sup>). The study received full Institutional Review Board (IRB) review and approval; all participants provided written informed consent prior to performing study procedures.

### Procedures

Participants who responded to media advertisements for persons interested in treatment studies for eating/weight concerns were evaluated by trained and monitored doctoral research clinicians to determine eligibility (ages 18-60 and meeting full DSM-IV criteria for BED, which exceeds DSM-5 criteria in behavioral frequency and duration stipulations). Individuals were excluded if they were receiving current treatments for eating/weight disorders, had medical conditions that could influence weight or eating (e.g., diabetes), had severe psychiatric illness that could interfere with assessment or required alternative treatments (e.g., psychosis, bipolar disorder), or if they were pregnant.

Two semi-structured interviews were administered by doctoral-level clinicians to establish DSM-IV-defined BED diagnosis, to assess eating-disorder psychopathology, and to diagnose psychiatric and personality disorders. Clinical interviews included assessment of historical variables, including ages of onset of obesity, dieting, binge eating, and lifetime psychopathology. Height and weight were measured during the evaluation process and participants completed a battery of self-report inventories.

## Assessments

*Structured Clinical Interview for DSM-IV (SCID-IV<sup>48</sup>)*. The SCID-IV, a structured diagnostic interview, was used to assess current and lifetime DSM-IV-defined psychiatric disorders, including BED. Kappa coefficients for interrater reliability for psychiatric disorders ranged from 0.68 to 1.0; kappa for BED was 1.0.

*Eating Disorder Examination Interview (EDE<sup>49</sup>)*. The EDE, a semi-structured investigator-based clinical interview<sup>50</sup> with demonstrated reliability for BED,<sup>51</sup> was used to confirm the SCID-IV-informed diagnosis of BED and to assess the features of eating-disorder psychopathology. The EDE assesses the frequency of objective binge episodes (OBE), defined as consuming objectively large quantities of food in a short period of time while experiencing a subjective loss of control over eating. In addition, the EDE assesses cognitive and behavioral features of eating psychopathology and generates four subscales (restraint, shape concern, weight concern, and eating concern) and a total “global” scale using a 7-point scale (0-6), where higher total scores reflect greater severity. In the present study, interrater reliability for eating psychopathology and binge-eating frequency was excellent (Spearman rho = 0.87-0.97 for the subscales and 0.99 for OBEs).

*Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV<sup>52</sup>)*. The DIPD-IV, a semi-structured diagnostic interview, was administered to assess for DSM-IV-defined personality disorders. The DIPD-IV guidelines require that a criterion be judged present and pervasive for at least two years and that it be viewed as characteristic of the individual throughout their adulthood. For this study, participants were categorized as having features of a personality disorder if they were one criterion short of meeting full diagnostic threshold given the conservative nature of the DIPD-IV. Kappa coefficients for interrater reliability for diagnoses ranged from 0.58 to 1.0.

The DIPD-IV includes a criterion assessing “impulsivity” in at least two domains. Participants were asked about patterns of engagement in 12 impulsive behaviors, including impulsive binge eating, sexual behavior, substance abuse, self-harm, and antisocial behaviors. This criterion was used to indicated clinically-elevated impulsivity, as in previous work.<sup>53</sup>

*Impulsivity Control Scale (ICS<sup>54,55</sup>)*. ICS is a 15-item self-report measure of impulsivity that is independent of aggressive behavior. ICS is related to UPPS-P dimensions of negative urgency, sensation seeking, and positive urgency.<sup>56</sup> The ICS was completed by a subsample of participants ( $N = 128$ ) and was used in this study as a “concurrent validity” check for the impulsivity criterion determined by the DIPD-IV interview.

*Beck Depression Inventory (BDI)* is a well-established 21-item measure of depressive symptoms.<sup>57,58</sup>

*Rosenberg Self-Esteem Scale (RSES)* is a 10-item measure of global self-esteem.<sup>59</sup>

## Analyses

Based on assessment of impulsivity via DIPD-IV, participants were categorized into two groups: (1) Individuals with BED and general impulsivity (GI+) and (2) Individuals with BED and without general impulsivity (GI-). The general impulsivity group had significantly higher scores on the ICS ( $t_{(126)} = 3.29$ ,  $P = .002$ ), supporting the concurrent validity of subgrouping based on the DIPD-IV interview assessment.

We investigated group differences in demographic features, developmental history, and psychological features, and group

differences in rates of comorbidity. Demographic features included age, sex, body mass index (BMI), and race/ethnicity. Developmental history included age of onset of obesity, binge eating, dieting, and history of lifetime psychiatric and personality disorder comorbidity. Psychological features included self-report measures of depressive symptoms and self-esteem (BDI and RSES) and measures of eating-disorder psychopathology (EDE interview). Groups were also compared on the frequency of current and lifetime psychiatric and personality disorders.

## Results

Participants were grouped based on the presence or absence of general impulsivity as assessed by the DIPD-IV. Of the 343 included participants with BED, 21% were categorized with general impulsivity (GI+;  $N = 73$ ) and 79% without general impulsivity (GI-;  $N = 270$ ).

### Demographic, developmental, psychological, and eating-disorder characteristics

Table 1 summarizes the characteristics and comparisons of the two BED groups. The BED groups with and without general impulsivity did not differ significantly in demographic characteristics, including age, sex, or race, or in BMI. The two groups did not differ significantly in age of onset of obesity, binge eating, or dieting. Individuals with general impulsivity and BED reported a

significantly greater number of lifetime psychiatric diagnoses than those without general impulsivity. Individuals with general impulsivity reported significantly greater levels of depressive symptoms but did not differ on self-esteem levels.

Individuals with general impulsivity reported significantly greater overall eating-disorder psychopathology (EDE-Global) with higher scores on three of the four EDE subscales (dietary restraint, shape concern, and weight concern, but not eating concern) compared to individuals without general impulsivity. The two groups did not differ significantly in frequency of binge eating.

Because the BED groups categorized with and without general impulsivity differed significantly in BDI scores and because higher levels of depressive/negative affect are associated with severity of BED,<sup>39</sup> subsequent analysis of co-variance (ANCOVA) examined group differences after statistically adjusting for BDI scores. Table 2 shows estimated marginal means after statistically adjusting for BDI and results from ANCOVA analyses. Even after adjusting for BDI scores, individuals categorized with general impulsivity still had significantly greater impulsivity levels (ICS), supporting the concurrent validity of DIPD-IV-based grouping. ANCOVAs (adjusting for BDI) revealed, consistent with ANOVA findings, that individuals with general impulsivity reported a significantly greater number of lifetime psychiatric diagnoses. ANCOVAs (adjusting for BDI) revealed that individuals with general impulsivity reported greater weight concern but differences on the other EDE scales no longer attained statistical significance.

**Table 1.** Demographic, Developmental, Psychological, and Eating-Disorder Characteristics of BED Patients With ( $N=73$ ) and Without ( $N=270$ ) General Impulsivity

	Measure	GI+		GI-		Statistics	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>t</i> / $\chi^2$	<i>P</i>
Demographic	Age ( <i>M, SD</i> )	43.86	9.74	44.93	9.13	0.87	.39
	BMI ( <i>M, SD</i> )	37.65	8.40	37.08	7.05	0.59	.56
	Sex (% female)	72.6%		75.6%		0.27	.61
	Race (% Caucasian)	76.7%		81.5%		4.29	.51
Developmental	Age first overweight	15.48	9.93	15.93	9.51	-0.35	.73
	Age of dieting onset	18.25	9.71	18.71	8.50	-0.39	.70
	Age first binge eating	23.81	12.31	22.59	11.80	0.75	.46
	Age of BED onset	26.85	12.35	25.56	12.74	0.75	.46
	# Lifetime diagnoses	1.93	1.47	1.37	1.23	<b>3.32</b>	<b>.001</b>
	Lifetime comorbidity (2+)	1.40	0.70	1.11	0.81	<b>2.72</b>	<b>.007</b>
Psychological measures	ICS	24.06	4.93	21.07	3.69	<b>3.29</b>	<b>.002</b>
	BDI	19.62	9.77	16.02	8.37	<b>3.14</b>	<b>.002</b>
	RSES	27.53	5.57	28.72	5.95	-1.54	.13
	EDE-Total	2.98	0.94	2.65	0.97	<b>2.43</b>	<b>.02</b>
	EDE-Restraint	2.15	1.27	1.80	1.30	<b>2.00</b>	<b>.045</b>
	EDE-Shape	3.95	1.23	3.63	1.12	<b>2.06</b>	<b>.04</b>
Eating-disorder psychopathology	EDE-Weight	3.56	1.01	3.11	0.98	<b>3.28</b>	<b>.001</b>
	EDE-Eating	2.25	1.36	2.08	1.85	0.67	.50
	OBEs	15.54	8.36	17.35	10.21	-0.86	.39

Individuals with general impulsivity (GI+) reported greater eating psychopathology (EDE-Global, EDE-Restraint, EDE-Shape, EDE-Weight) than those without general impulsivity (GI-). There were no differences in number of objective binge episodes (OBEs) or eating concern (EDE-Eating). Individuals with general impulsivity reported greater depressive symptoms and lifetime psychopathology. **Bold** indicates significant results.

Abbreviations: BDI, Beck Depression Inventory; BMI, body mass index; ICS, Impulsivity Control Scale; *M*, mean; RSES, Rosenberg Self Esteem Scale; *SD*, standard deviation.

**Table 2.** Demographic, Developmental, Psychological, and Eating-Disorder Characteristics of BED Patients With ( $N=73$ ) and Without ( $N=270$ ) General Impulsivity, After Statistically Adjusting for BDI

	Measure	GI+		GI–		Statistics	
		EMM	SE	EMM	SE	F	P
<i>Demographic</i>	BMI	38.47	1.41	35.68	0.88	2.78	.10
	Age first overweight	15.96	1.27	15.94	0.66	0.01	.99
	Age of dieting onset	18.80	1.17	18.31	0.61	0.13	.72
	Age first binge eating	23.52	1.55	22.04	0.80	0.71	.40
	Age of BED onset	26.73	1.58	25.17	0.86	0.67	.41
	# Lifetime diagnoses	1.80	0.14	1.41	0.07	<b>5.72</b>	<b>.02</b>
<i>Developmental</i>	Lifetime comorbidity (2+)	1.31	0.09	1.14	0.05	2.90	.09
	ICS	23.67	0.66	21.20	0.41	<b>9.96</b>	<b>.002</b>
<i>Psychological measures</i>	RSES	28.50	0.73	27.31	0.45	1.91	.17
	EDE-Total	2.84	0.11	2.69	0.05	1.48	.23
	EDE-Restraint	2.10	0.16	1.81	0.08	2.70	.10
	EDE-Shape	3.78	0.13	3.67	0.06	0.58	.45
	EDE-Weight	3.42	0.11	3.15	0.06	<b>4.78</b>	<b>.03</b>
	EDE-Eating	2.05	0.21	2.14	0.10	0.15	.70
<i>Eating-disorder psychopathology</i>	OBEs	15.15	1.22	17.45	0.61	1.42	.23

Estimated marginal means (EMM), standard error (SE), and ANCOVA results are presented in this table. When statistically adjusting for depressive symptoms, individuals with BED and general impulsivity (GI+) had significantly greater impulsivity, different numbers of lifetime psychological diagnoses, and greater weight concern than individuals without general impulsivity (GI–).

**Bold indicates significant results.**

Abbreviations: BDI, Beck Depression Inventory; BMI, body mass index; EDE, Eating Disorder Examination; ICS, Impulsivity Control Scale; OBE, objective binge episode; RSES, Rosenberg Self Esteem Scale.

### Rates of comorbidity

Table 3 summarizes rates of diagnostic comorbidity. Patients categorized with general impulsivity had significantly higher rates of psychiatric and personality disorder comorbidity than those without general impulsivity. Specifically, they had significant higher rates of current diagnoses (i.e., major depressive disorder [MDD], generalized anxiety disorder [GAD], substance abuse, drug abuse, and alcohol abuse) and lifetime diagnoses (i.e., substance abuse, drug abuse, and bulimia nervosa). Additionally, they had higher rates of personality disorders, including cluster B (e.g., antisocial personality disorder and borderline personality disorder) and cluster C diagnoses (e.g., avoidant personality disorder and obsessive-compulsive personality disorder).

### Discussion

This study investigated whether individuals with BED and general impulsivity differ in clinical presentation from those without general impulsivity. Similar to findings for individuals with “multi-impulsive” bulimia nervosa, individuals with BED and general impulsivity reported greater eating-related psychopathology, greater depressive symptoms, and greater rates of comorbidity than those without general impulsivity. Thus, a general-impulsive subtype could signal a more severe variant of BED characterized by heightened and broader psychopathology.

Given the unknown nature of impulse control difficulties in BED, the frequency of general impulsivity in this sample is notable. In line with research suggesting that impulsivity in BED can be food-specific,<sup>11–13,17</sup> 79% of this sample reported impulse control problems only in the context of food. However, 21% met clinical criteria for impulsivity in multiple domains. For comparison, data

from the National Comorbidity Replication survey found that 43.3% of persons with BED reported comorbid impulse control disorders and 23.3% reported comorbid substance use disorders.<sup>31</sup> Because of divergent findings about different forms of impulsivity in BED, future work should examine whether individuals with BED and general impulsivity perform differently than those without general impulsivity on behavioral, neurocognitive, and brain-based measures of both food-specific and general impulse control.

Compared to individuals without general impulsivity, individuals with BED plus general impulsivity had a more severe clinical presentations in both eating-disorder feature domains as well as with respect to heightened rates of several psychiatric and personality disorders. For example, they reported greater lifetime rates of alcohol use and drug use disorders, in line with a broader literature<sup>1,31</sup> and research on potential shared neurobiological features between BED and these conditions.<sup>13,36–38</sup> Consistent with prior work investigating depressive/negative affect and comorbidity-based subtyping of BED,<sup>39,40</sup> individuals with BED and general impulsivity reported both greater depressive/negative affect and higher rates of comorbidity. Importantly, even after statistically adjusting for current depressive symptoms, group differences in impulsivity, lifetime comorbidity, and weight-related psychopathology remained significant. Collectively, these findings suggest continued efforts are needed by risk-factor, developmental, and experimental studies to examine how depressive/negative affect, comorbidity, and impulsivity-related factors interface to shape the course and outcome of BED.

The present work converges with older work investigating “multi-impulsivity” in bulimia nervosa. In this study, individuals with BED and general impulsivity had a greater lifetime history of bulimia nervosa, suggesting shared impulsivity-related risk for these conditions.<sup>43</sup> Prior work found that individuals with “multi-impulsive”

**Table 3.** Psychiatric and Personality Diagnoses in BED Patients With ( $N=73$ ) and Without ( $N=270$ ) General Impulsivity

		GI+	GI–	Statistics		
		%	%	$\chi^2$	$P$	$N_{d/o}$
<i>Current</i>	Any mood d/o	60.3%	52.6%	1.37	.24	186
	Any anxiety d/o	15.1%	14.1%	0.05	.83	49
	MDD	27.8%	15.8%	<b>5.44</b>	<b>.02</b>	62
	GAD	18.1%	6.7%	<b>8.95</b>	<b>.003</b>	31
	PTSD	4.3%	3.4%	0.12	.73	12
	Substance abuse	11.0%	0.7%	<b>21.29</b>	<b>&lt;.001</b>	10
	Drug abuse	4.1%	0.4%	<b>6.97</b>	<b>.008</b>	4
<i>Psychiatric diagnoses</i>	Alcohol abuse	6.8%	0.4%	<b>14.04</b>	<b>&lt;.001</b>	6
<i>Lifetime</i>	Any mood d/o	40.8%	38.2%	0.12	.73	99
	Any anxiety d/o	17.6%	19.6%	0.10	.75	50
	MDD	28.8%	27.8%	0.03	.87	96
	PTSD	4.1%	2.0%	0.47	.49	10
	Substance abuse	38.5%	21.6%	<b>7.91</b>	<b>.005</b>	83
	Drug abuse	27.1%	12.3%	<b>9.46</b>	<b>.002</b>	52
	Alcohol abuse	27.9%	19.3%	2.42	.12	71
	Bulimia nervosa	11.0%	4.4%	<b>4.44</b>	<b>.04</b>	20
<i>Psychiatric diagnoses</i>	Anorexia nervosa	2.7%	0.7%	1.99	.16	4
<i>Personality diagnoses</i>	Any personality d/o	57.5%	37.4%	<b>13.47</b>	<b>.001</b>	143
	Cluster A	12.3%	6.3%	2.99	.08	26
	Cluster B	23.3%	4.4%	<b>26.36</b>	<b>&lt;.001</b>	29
	Cluster C	47.9%	30.4%	<b>7.90</b>	<b>.005</b>	117

Individuals with BED and general impulsivity (GI+) had greater rates of psychiatric and personality diagnoses than individuals without general impulsivity (GI–). Features and diagnosis of personality psychopathology included.  $N=0$  reported lifetime GAD without current d/o. **Bold** indicates significant results. Abbreviations: d/o, disorder; GAD, generalized anxiety disorder; MDD, major depressive disorder;  $N_{d/o}$ ,  $N$  who meet criteria for disorder; PTSD, post-traumatic stress disorder.

bulimia nervosa had more severe eating psychopathology, greater depressive symptoms, greater rates of comorbidity, and poorer response to treatment.<sup>41–45,60</sup> Interestingly, as in the present study, individuals with “multi-impulsive” bulimia nervosa had greater reported psychopathology without differences in frequency of binge eating.<sup>42,44,60</sup> Further, individuals with “multi-impulsive” bulimia nervosa had worse treatment outcomes and seemed to exhibit “behavioral substitution” during eating disorder treatment.<sup>46</sup> Accordingly, parallels between “multi-impulsive” bulimia nervosa and general impulsivity in BED may have important clinical and treatment implications.

Why might general impulsivity be associated with increased rates of comorbid psychopathology in BED? Shared reinforcement patterns across impulsive behaviors (e.g., binge eating, substance use, self-harm, risky sexual behavior) could contribute to the development of multiple psychological conditions. For example, negative urgency—or a tendency to act rashly when distressed—offers a greater risk for psychopathology across disorders<sup>61</sup> and is associated with poorer eating-disorder treatment outcomes.<sup>62</sup> Similarly, positive urgency—or a tendency to engage in impulsive behaviors due to positive emotion—is associated with impulse control-related psychopathology, including substance use disorders and eating disorders.<sup>63</sup> Individuals with greater intensity of negative or positive affect may be more susceptible to affectively-driven impulsive behaviors across domains. These negative and positive reinforcement patterns are consistent with neurobiological

models of shared mechanisms across eating and substance use, functional assessment of binge eating/purging, and recent work in samples with comorbid binge eating and non-suicidal self-injury.<sup>13,37,38,64,65</sup>

This study has several strengths, including its large sample size and the use of semi-structured interviews reliably administered by trained doctoral research clinicians to assess clinically-significant impulsive behaviors and psychopathology. Further, because the DIPD-IV investigates impulsivity across many domains (eg, substance use, risky sexual behavior, and antisocial behavior), our categorization of general impulsivity included clinically-significant impulsive behaviors across a wider scope than prior work in “multi-impulsive” BN that was primarily defined based on self-harm and/or comorbid substance use.<sup>44</sup>

The study’s findings, however, should be understood in the contexts of its limitations. First, the sample included in this paper was defined based on DSM-IV criteria rather than DSM-5 criteria and therefore it is possible that the patient group in this study may be characterized by more frequent binge eating and/or more severe psychopathology than DSM-5-based studies. The majority of participants were female, Caucasian, married, and college-educated, which may limit generalizability to more diverse groups or to groups with different characteristics. Participants were seeking treatment at a university-based program and therefore the findings may not be generalizable to community or nontreatment-seeking groups. There was no assessment of food-related obsessions/

compulsions or planned binge eating in this study. Future work should examine the links of impulsivity and compulsivity with co-morbidity, symptom severity, and treatment outcomes for patients with BED.

Research is needed to determine whether adapting or enhancing treatment for BED to address general impulsivity may influence treatment outcomes. Recent food impulsivity-targeted treatments<sup>18,19</sup> may or may not be suited to individuals without general impulsivity. In contrast, broader-impulsivity targeted treatments may be helpful for individuals with BED and general impulsivity. General skills training (e.g., for cognitive behavioral therapy: urge surfing applied to alcohol and food, cognitive reappraisal practiced in multiple contexts) could prevent “behavioral substitution” and improve treatment outcomes. Psychological treatments that address impulsivity and interpersonal features, such as dialectical behavior therapy<sup>66</sup> and interpersonal psychotherapy,<sup>67,68</sup> might be especially well-suited for patients with BED who also have general impulsivity. Pharmacological agents that treat BED and reduce impulsivity symptoms, including lisdexamfetamine dimesylate (LDX), may be especially effective in individuals with difficulties related to general impulsivity.<sup>4,69,70</sup> Treatment trials for BED may benefit from investigations of general impulsivity and its effect on treatment outcomes.

## Conclusions

In sum, subtyping within BED based on general impulsivity identifies a group with more severe clinical presentation. These individuals reported greater eating-related psychopathology, greater comorbid and lifetime history of psychopathology, and greater depressive symptoms. Building upon older investigations of “multi-impulsive” bulimia nervosa, this work suggests that adapting and adding interventions based on general impulsivity for BED may have utility.

**Financial Support.** This research was supported, in part, by National Institutes of Health grants R01 DK49587 and R01 DK121551 (Grilo). Dr. Grilo was also supported, in part, by NIH grants R01 DK114075 and R01 DK112771. Funders played no role in the content of this paper.

**Disclosures.** Dr. Rebecca Boswell and Dr. Carlos Grilo do not have any conflicts of interest. Dr. Grilo reports several broader interests which did not influence this research or paper. Dr. Grilo’s broader interests include: Consultant to Sunovion and Weight Watchers; Honoraria for lectures, CME activities, and presentations at scientific conferences and Royalties from Guilford Press and Taylor & Francis Publishers for academic books.

## References

1. Udo T, Grilo CM. Prevalence and correlates of DSM-5-defined eating disorders in a nationally representative sample of U.S. adults. *Biol Psychiatry*. 2018;**84**(5):345–354.
2. Udo T, Bitley S, Grilo CM. Suicide attempts in U.S. adults with lifetime DSM-5 eating disorders. *BMC Med*. 2019;**17**:120
3. APA. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
4. Citrome L. Binge eating disorder revisited: what’s new, what’s different, what’s next. *CNS Spectr*. 2019;**24**(S1):4–13.
5. Dawe S, Loxton NJ. The role of impulsivity in the development of substance use and eating disorders. *Neurosci Biobehav Rev*. 2004;**28**(3):343–351.
6. Robbins TW, Gillan CM, Smith DG, de Wit S, Ersche KD. Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends Cogn Sci*. 2012;**16**(1):81–91.
7. Deal LS, Wirth RJ, Gasior M, Herman BK, McElroy SL. Validation of the Yale-Brown Obsessive Compulsive Scale modified for binge eating. *Int J Eat Disord*. 2015;**48**(7):994–1004.
8. Wonderlich SA, Connolly KM, Stice E. Impulsivity as a risk factor for eating disorder behavior: assessment implications with adolescents. *Int J Eat Disord*. 2004;**36**(2):172–182.
9. Pearson CM, Zapolski TC, Smith GT. A longitudinal test of impulsivity and depression pathways to early binge eating onset. *Int J Eat Disord*. 2015;**48**(2):230–237.
10. Manasse SM, Espel HM, Schumacher LM, et al. Does impulsivity predict outcome in treatment for binge eating disorder? A multimodal investigation. *Appetite*. 2016;**105**:172–179.
11. Giel KE, Teufel M, Junne F, Zipfel S, Schag K. Food-related impulsivity in obesity and binge eating disorder—a systematic update of the evidence. *Nutrients*. 2017;**9**:1170
12. Schag K, Schonleber J, Teufel M, Zipfel S, Giel KE. Food-related impulsivity in obesity and binge eating disorder—a systematic review. *Obes Rev*. 2013;**14**(6):477–495.
13. Balodis IM, Grilo CM, Potenza MN. Neurobiological features of binge eating disorder. *CNS Spectr*. 2015;**20**:557–565.
14. Hege MA, Stingl KT, Kullmann S, et al. Attentional impulsivity in binge eating disorder modulates response inhibition performance and frontal brain networks. *Int J Obes (Lond)*. 2015;**39**(2):353–360.
15. Svaldi J, Naumann E, Trentowska M, Schmitz F. General and food-specific inhibitory deficits in binge eating disorder. *Int J Eat Disord*. 2014;**47**(5):534–542.
16. Kober H, Boswell RG. Potential psychological & neural mechanisms in binge eating disorder: implications for treatment. *Clin Psychol Rev*. 2018;**60**:32–44.
17. Voon V. Cognitive biases in binge eating disorder: the hijacking of decision making. *CNS Spectr*. 2015;**20**(6):566–573.
18. Boutelle KN, Knatz S, Carlson J, Bergmann K, Peterson CB. An open trial targeting food cue reactivity and satiety sensitivity in overweight and obese binge eaters. *Cogn Behav Pract*. 2017;**24**:363–373.
19. Schag K, Rennhak SK, Leehr EJ, et al. IMPULS: impulsivity-focused group intervention to reduce binge eating episodes in patients with binge eating disorder—a randomised controlled trial. *Psychother Psychosom*. 2019;**88**(3):141–153.
20. Calero-Elvira A, Krug I, Davis K, Lopez C, Fernandez-Aranda F, Treasure J. Meta-analysis on drugs in people with eating disorders. *Eur Eat Disord Rev*. 2009;**17**(4):243–259.
21. Stein D, Lilienfeld LR, Wildman PC, Marcus MD. Attempted suicide and self-injury in patients diagnosed with eating disorders. *Compr Psychiatry*. 2004;**45**(6):447–451.
22. Goldner EM, Geller J, Birmingham CL, Remick RA. Comparison of shoplifting behaviours in patients with eating disorders, psychiatric control subjects, and undergraduate control subjects. *Can J Psychiatry*. 2000;**45**:471–475.
23. Culbert KM, Klump KL. Impulsivity as an underlying factor in the relationship between disordered eating and sexual behavior. *Int J Eat Disord*. 2005;**38**(4):361–366.
24. Balodis IM, Kober H, Worhunsky PD, et al. Monetary reward processing in obese individuals with and without binge eating disorder. *Biol Psychiatry*. 2013;**73**(9):877–886.
25. Balodis IM, Molina ND, Kober H, et al. Divergent neural substrates of inhibitory control in binge eating disorder relative to other manifestations of obesity. *Obesity (Silver Spring)*. 2013;**21**(2):367–377.
26. Svaldi J, Brand M, Tuschen-Caffier B. Decision-making impairments in women with binge eating disorder. *Appetite*. 2010;**54**(1):84–92.
27. Manasse SM, Goldstein SP, Wyckoff E, et al. Slowing down and taking a second look: Inhibitory deficits associated with binge eating are not food-specific. *Appetite*. 2016;**96**:555–559.
28. Davis C, Levitan RD, Carter J, et al. Personality and eating behaviors: a case-control study of binge eating disorder. *Int J Eat Disord*. 2008;**41**(3):243–250.

29. Udo T, Grilo CM. Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. *Int J Eat Disord*. 2019;**52**:42–50.
30. Becker DF, Grilo CM. Comorbidity of mood and substance use disorders in patients with binge-eating disorder: associations with personality disorder and eating disorder pathology. *J Psychosom Res*. 2015;**79**(2):159–164.
31. Hudson JI, Hiripi E, Pope HG, Kessler RM. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry*. 2007;**61**(3):348–358.
32. Kessler RC, Berglund PA, Chiu WT, *et al.* The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biol Psychiatry*. 2013;**73**(9):904–914.
33. Grilo CM, White MA, Masheb RM. DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. *Int J Eat Disord*. 2009;**42**(3):228–234.
34. Javaras KN, Pope HG, Lalonde JK, *et al.* Co-occurrence of binge eating disorder with psychiatric and medical disorders. *J Clin Psychiatry*. 2008;**69**(2):266–273.
35. Friborg O, Martinussen M, Kaiser S, *et al.* Personality disorders in eating disorder not otherwise specified and binge eating disorder: a meta-analysis of comorbidity studies. *J Nerv Ment Dis*. 2014;**202**(2):119–125.
36. Schulte EM, Grilo CM, Gearhardt AN. Shared and unique mechanisms underlying binge eating disorder and addictive disorders. *Clin Psychol Rev*. 2016;**44**:125–139.
37. Kessler RM, Hutson PH, Herman BK, Potenza MN. The neurobiological basis of binge-eating disorder. *Neurosci Biobehav Rev*. 2016;**63**:223–238.
38. Davis C. The epidemiology and genetics of binge eating disorder (BED). *CNS Spectr*. 2015;**20**(6):522–529.
39. Grilo CM, Masheb RM, Wilson GT. Subtyping binge eating disorder. *J Consult Clin Psychol*. 2001;**69**(6):1066–1072.
40. Peterson CB, Miller KB, Crow SJ, Thuras P, Mitchell JE. Subtypes of binge eating disorder based on psychiatric history. *Int J Eat Disord*. 2005;**38**:273–276.
41. Lacey JH, Moureli E. Bulimic alcoholics: some features of a clinical subgroup. *Br J Addict*. 1986;**81**:389–393.
42. Wiederman MW, Pryor T. Multi-impulsivity among women with bulimia nervosa. *Int J Eat Disord*. 1996;**20**(4):359–365.
43. Waxman SE. A systematic review of impulsivity in eating disorders. *Eur Eat Disord Rev*. 2009;**17**:408–425.
44. Bell L, Newsn K. What is multi-impulsive bulimia and can multi-impulsive patients benefit from supervised self-help? *Eur Eat Disord Rev*. 2002;**10**(6):413–427.
45. Fichter MM, Quadfleig N, Rief W. Course of multi-impulsive bulimia. *Psychol Med*. 1994;**24**:591–604.
46. Brisman J, Siegel M. Bulimia and alcoholism: two sides of the same coin? *J Subst Abuse Treat*. 1984;**1**(2): 113–118.
47. Becker DF, Masheb RM, White MA, Grilo CM. Psychiatric, behavioral, and attitudinal correlates of avoidant and obsessive-compulsive personality pathology in patients with binge-eating disorder. *Compr Psychiatry*. 2010;**51**(5):531–537.
48. First MB, Gibbon M, Spitzer RL, Williams JBW, Benjamin LS. *Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I)*. New York, NY: New York State Psychiatric Institute, Biometrics Research Department; 1996.
49. Fairburn CG, Cooper Z. The eating disorder examination (12th edition). In: FCG, Wilson GT, eds. *Binge Eating: Nature, Assessment, and Treatment*. New York, NY: Guilford Press; 1993:317–360.
50. Grilo CM, Masheb RM, Wilson GT. A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. *J Consult Clin Psychol*. 2001;**69**:317–322.
51. Grilo CM, Masheb RM, Lozano-Blanco C, Barry DT. Reliability of the Eating Disorder Examination in patients with binge eating disorder. *Int J Eat Disord*. 2004;**35**:80–85.
52. Zanarini MC, Frankenburg FR, Sickel AE, Yong L. *The Diagnostic Interview for DSM-IV Personality Disorders*. Vol. 340. Belmont, MA: McLean Hospital; 1996.
53. Weiss NH, Tull MT, Viana AG, Anestis MD, Gratz KL. Impulsive behaviors as an emotion regulation strategy: examining associations between PTSD, emotion dysregulation, and impulsive behaviors among substance dependent inpatients. *J Anxiety Disorder*. 2012;**26**:453–458.
54. Plutchik R, van Praag HM. The measurement of suicidality, aggressivity, and impulsivity. *Prog Neuropsychopharmacol Biol Psychiatry*. 1989;**13**: 523–534.
55. Grosz DE, Lipschitz DS, Sofia E, *et al.* Correlates of violence risk in hospitalized adolescents. *Compr Psychiatry*. 1994;**35**:296–300.
56. Verdejo-Garcia A, Lozano O, Moya M, Alcazar MA, Perez-Garcia M. Psychometric properties of a Spanish version of the UPPS-P Impulsive Behavior Scale: reliability, validity, and association with trait and cognitive impulsivity. *J Pers Assess*. 2010;**92**(1):70–77.
57. Beck AT, Steer R. *Manual for Revised Beck Depression Inventory*. New York, NY: Psychological Corporation; 1987.
58. Beck AT, Steer R, Garbin M. Psychometric properties of the Beck Depression Inventory: 25 years of evaluation. *Clin Psychol Rev*. 1998;**8**:77–100.
59. Rosenberg M. *Society and the Adolescent Self-Image*. Princeton, NJ: Princeton University Press; 1965.
60. Myers TC, Wonderlich SA, Crosby R, *et al.* Is multi-impulsive bulimia a distinct type of bulimia nervosa: psychopathology and EMA findings. *Int J Eat Disord*. 2006;**39**(8):655–661.
61. Berg JM, Latzman RD, Bliswise NG, Lilienfeld LR. Parsing the heterogeneity of impulsivity: a meta-analytic review of the behavioral implications of the UPPS for psychopathology. *Psychol Assess*. 2015;**27**(4):1129–1146.
62. Bardone-Cone AM, Butler RM, Balk MR, Koller KA. Dimensions of impulsivity in relation to eating disorder recovery. *Int J Eat Disord*. 2016;**49**(11):1027–1031.
63. Smith GT, Cyders MA. Integrating affect and impulsivity: the role of positive and negative urgency in substance use risk. *Drug Alcohol Depend*. 2016;**162**:S3–S12.
64. Muehlenkamp JJ, Takakuni S, Brausch AM, Peyrel N. Behavioral functions underlying NSSI and eating disorder behaviors. *J Clin Psychol*. 2019;**75**(7): 1219–1232.
65. Wedig MM, Nock MK. The functional assessment of maladaptive behaviors: a preliminary evaluation of binge eating and purging among women. *Psychiatry Res*. 2010;**178**(3):518–524.
66. Telch TF, Agras WS, Linehan MM. Dialectical behavior therapy for binge eating disorder. *J Consult Clin Psychol*. 2001;**69**:1061–1065.
67. Markowitz JG. Interpersonal therapy of personality disorders. In: Oldham JM, Skodol AE, Bender BS, eds. *Textbook of Personality Disorders*. Washington, DC: American Psychiatric Press; 2005:321–334.
68. Tanofsky-Kraff M, Wilfley DE. Interpersonal psychotherapy for bulimia nervosa and binge-eating disorder. In: Grilo CM, Mitchell JE, eds. *The Treatment of Eating Disorders: A Clinical Handbook*. New York, NY: The Guilford Press; 2009:271–293.
69. McElroy SL, Mitchell JE, Wilfley D, *et al.* Lisdexamfetamine dimesylate effects on binge eating behavior and obsessive-compulsive and impulsive features in adults with binge eating disorder. *Eur Eat Disord Rev*. 2016;**24**: 223–231.
70. McElroy SL, Guerdjikova AI, Mori N, Munoz MR, Keck PE. Overview of the treatment of binge eating disorder. *CNS Spectr*. 2015;**20**(6):546–556.