

Clinical Features and Physiological Response to a Test Meal in Purging Disorder and Bulimia Nervosa

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Context: Recent data suggest that purging disorder, a recently characterized form of eating disorder not otherwise specified, may be worthy of specific delineation in nosological schemes. However, more data are needed to determine how purging disorder differs from bulimia nervosa.

Objective: To examine clinical features and subjective as well as objective physiological responses to a standardized test meal in purging disorder compared with bulimia nervosa and controls.

Design: Study visit 1 included psychological assessments with structured clinical interviews and questionnaires. Study visit 2 included assessment of test-meal responses.

Setting: Participants recruited from the community completed test-meal studies in a General Clinical Research Center.

Participants: Women with DSM-IV bulimia nervosa-purging subtype (n=37) and purging disorder (n=20) and non-eating disorder controls (n=33) with a body mass index (calculated as weight in kilograms divided by height in meters squared) between 18.5 and 26.5 who were free of psychotropic medications.

Main Outcome Measures: Assessments of eating disorder severity, postprandial cholecystokinin response, and subjective responses to test meals.

Results: Eating abnormalities were significantly elevated in participants with purging disorder and bulimia nervosa compared with controls but did not differ between eating disorder groups. Participants with purging disorder demonstrated significantly greater postprandial cholecystokinin release compared with participants with bulimia nervosa ($t_{76,44}=2.51$; $P=.01$) and did not differ significantly from controls ($t_{75,93}=0.03$; $P=.98$). Participants with purging disorder reported significantly greater postprandial fullness and gastrointestinal distress compared with participants with bulimia nervosa and controls.

Conclusions: Purging disorder is a clinically significant disorder of eating that appears to be distinct from bulimia nervosa on subjective and physiological responses to a test meal. Findings support further consideration of purging disorder for inclusion in the classification of eating disorders. Future studies on the psychobiology of purging disorder are needed to understand the propensity to purge in the absence of binge eating.

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PURGING DISORDER (PD) IS A recently described form of eating disorder not otherwise specified that is characterized by recurrent purging (eg, self-induced vomiting) to control weight or shape among normal-weight individuals in the absence of binge eating.¹ Individuals with PD may report a loss of control over their eating, but they do not consume more food than most people would under similar circumstances. Thus, PD does not involve binge eating as defined by the DSM-IV. Further, some individuals with PD report no loss of control over their eating. Instead, purging occurs following regular meals or snacks.

Several studies support the clinical significance of PD,¹⁻⁴ and a recent epidemiological study in Australia found that PD was

the most prevalent form of eating disorder, affecting more women than anorexia nervosa (AN) and bulimia nervosa (BN) combined.⁵ Furthermore, PD has demonstrated longitudinal stability and distinctiveness from BN on measures related to appetite regulation.¹ Specifically, women with PD reported lower hunger and less disinhibition around food compared with women with BN. These differences were maintained at 6-month follow-up, as was diagnostic status.¹ These findings led us to posit that PD and BN may be characterized by differences in physiological mechanisms that influence food intake, such as cholecystokinin (CCK) function.

Cholecystokinin is released from the upper tract of the small intestine in response to food intake. Experimental administration of CCK causes cessation of

food intake and the behavioral sequence of satiety in animals⁶⁻⁸ as well as subjective reports of satiation in humans.^{9,10} Women with BN have demonstrated blunted postprandial CCK response compared with healthy controls.¹¹⁻¹³ These differences may explain deficits in satiety, increased food intake found in feeding laboratory studies,¹⁴⁻¹⁷ and binge eating in BN.

Given the absence of binge eating in PD,¹ we posited that women with PD would differ from women with BN on CCK response to a standardized test meal but would not differ significantly from non-eating disorder controls. We further hypothesized that women with PD would report greater satiation in response to the test meal compared with women with BN but would not differ significantly from non-eating disorder controls. Finally, we sought to replicate previous findings regarding the clinical severity of PD^{1,2} as well as differences between PD and BN on measures of hunger and disinhibition.¹

METHOD

PARTICIPANTS

Women who met *DSM-IV* criteria for BN-purging subtype ($n=37$) and study criteria for PD (**Figure 1**) ($n=20$) and non-eating disorder controls ($n=33$) were recruited from the community in either the Boston, Massachusetts, ($n=45$, 50%) or Iowa City, Iowa, areas ($n=45$, 50%). (Although an even number of participants completed assessments in each location [ie, 45 participants were run at each site's General Clinical Research Center (GCRC)], 92% of participants completed study visit 1 psychological assessments in 1 of our laboratories [P.K.K.], which moved from Massachusetts to Iowa midway through the study, and the remaining 8% completed study visit 1 assessments at the Beth Israel Deaconess GCRC.) This represents a subset of a larger sample assessed for indicators of distress and disability associated with PD, results of which will be presented elsewhere. Advertisements on public transportation, in newspapers, and on posters at local college campuses invited women with no eating problems, women who binged and purged, and women who used "extreme measures to control weight" to call a toll-free number. Telephone screens were conducted for initial assessments of eligibility.

Participants were assessed with the Eating Disorder Examination (EDE)¹⁸ and Module H of the Structured Clinical Interview for *DSM-IV* Axis I Disorders (SCID-I)¹⁹ to confirm diagnostic group. Additional inclusion criteria for all participants were age between 18 and 45 years and body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) within a healthy range, between 18.5 and 26.5, based on objective height and weight assessments at study visit 1 (see "Procedures" subsection). Exclusion criteria were psychotropic medication use within 8 weeks of test-meal procedures; medical conditions or treatment that could influence appetite, weight, or ability to participate; current pregnancy or lactation; and history of moderate obesity ($BMI > 35$). For participants with PD, lifetime histories of *DSM-IV* BN or binge-eating disorder based on SCID-I were exclusion criteria. For non-eating disorder controls, lifetime history of eating disorder symptoms based on SCID-I, Three-Factor Eating Questionnaire²⁰ Cognitive Restraint Scores higher than 9, and dietary restriction for weight loss within the past 8 weeks based on self-report were exclusion criteria.

There were no significant differences among groups in age, race, or educational status. Mean (SD) age was 21.50 (3.96) years. Racial/ethnic composition of the sample, as endorsed by

1. Recurrent purging to influence weight or shape, such as self-induced vomiting, misuse of laxatives, diuretics, or enemas.
2. Purging occurs, on average, at least twice a week for 3 months.
3. Self-evaluation is unduly influenced by body shape or weight.
4. The purging is not associated with objectively large binge episodes.
5. The purging does not occur exclusively during the course of anorexia nervosa.

Figure 1. Study criteria for purging disorder.

participants, was 84.4% white, 3.3% Black/African American, 10% Asian/Pacific Islander, 1.1% Hispanic, and 1.1% undisclosed; 73.3% had high school, 24.5% had college, and 2.2% had graduate degrees.

Current rates of Axis I disorders were as follows: controls: major depressive disorder (MDD), 0%; anxiety disorder, 3%; and substance use disorder (SUD), 3%; BN: MDD, 16%; anxiety disorder, 19%; and SUD, 14%; and PD: MDD, 5%; anxiety disorder, 45%; and SUD, 20%. Lifetime histories of AN were found in 14% of participants with BN and 15% of participants with PD ($\chi^2=0.02$; $P=.88$; Fisher exact test $P>.99$). Participants with lifetime histories of AN had been weight recovered for at least 1 year (range, 22-96 months), and there was no significant difference between BN and PD in duration of weight recovery ($t_6=0.87$; $P=.42$).

PROCEDURES

Written informed consent was obtained from participants prior to participation, and this study received institutional review board approval. Procedures included 2 study visits. During study visit 1, participants completed clinical and height/weight assessments to establish eligibility. Participants also consumed the standardized test meal used in study visit 2 and made ratings to acclimate themselves to test-meal procedures. Participants were paid \$35 for study visit 1 and \$65 for study visit 2.

Study visit 2 was scheduled to occur at 8 AM at a GCRC after an overnight fast and within approximately 2 weeks of the first study visit (median time between visits [range] was 2.6 weeks [1 day to 4 months] [structured clinical interview assessment of diagnostic criteria was repeated prior to study visit 2 for any participant who completed study visit 2 more than 4 weeks after study visit 1 to ensure continued eligibility]). Participants were asked to consume nothing other than water after 11 PM the night before study visit 2. Participants completed a health screen to confirm adherence to the overnight fast and the absence of medications or medical conditions that would preclude participation in CCK response assessment. In addition, height, weight (reported in **Table 1**), and vital signs were measured by GCRC nursing staff. Following these assessments, an indwelling intravenous catheter was inserted in the participant's arm. Participants were given a 15-minute rest period and questionnaires to complete prior to blood draws. Baseline blood samples were drawn prior to the test meal. Participants were then given a liquid test meal to consume as quickly as comfortable over the course of 5 minutes. The test meal was a commercially available liquid supplement (Ensure Plus; Abbott Laboratories, Abbott Park, Illinois) consisting of 900 kcal in 600 g of fluid: 30% fat, 15% protein, and 55% carbohydrate. Blood samples were obtained 15 and 30 minutes following test-meal completion, corresponding to time to peak CCK release.¹¹⁻¹³ Prior to collection of blood samples, participants rated the following questions using a 100-mm visual analog scale (VAS): "satiated (satisfied)," "full," "hungry," "urge to binge," "urge to vomit," "tense," and "sad," anchored from "not at all/no" to "extremely," similar to methods used in previous studies.^{11,15-17} In addition, participants completed assessments of adverse effects on a 5-point Likert scale²⁴ at each assessment.

Table 1. Comparisons of Groups on Clinical Measures

Measure	Mean (SE)			F _{2,87}
	Control (n = 33)	Purging Disorder (n = 20)	Bulimia Nervosa (n = 37)	
Age, y	22.2 (0.7)	21.2 (0.9)	21.1 (0.7)	0.71
Height, cm	165.8 (1.1)	163.6 (1.4)	166.0 (1.0)	1.12
Weight, kg	61.5 (1.2)	59.5 (1.6)	61.5 (1.2)	0.52
BMI	22.5 (0.3)	22.1 (0.4)	22.3 (0.3)	0.12
EDE ¹⁸ interview				
Total	0.2 (0.1)*	3.5 (0.2)†	3.6 (0.1)†	226.66 ^a
Restraint	0.1 (0.2)*	3.6 (0.2)†	3.7 (0.2)†	123.20 ^a
Eating concern	0.0 (0.2)*	2.3 (0.3)†	2.7 (0.2)†	53.22 ^a
Weight concern	0.4 (0.2)*	4.1 (0.2)†	4.2 (0.2)†	180.43 ^a
Shape concern	0.3 (0.1)*	3.8 (0.2)†	4.0 (0.1)†	244.50 ^a
				t ₅₅ ^b
Vomit/wk	NA	5.5 (1.0)	5.6 (0.7)	-0.09
Purge/wk	NA	6.7 (1.1)	7.0 (0.8)	-0.18
Self-report				F _{2,87}
BSQ ²¹	43.2 (3.5)*	130.3 (4.5)†	137.7 (3.3)†	223.06 ^a
TFEQ ²⁰				
Cognitive restraint	2.6 (0.6)*	16.8 (0.8)†	15.9 (0.6)†	162.42 ^a
Disinhibition	3.3 (0.5)*	8.5 (0.6)†	13.2 (0.4)‡	127.64 ^a
Hunger	3.5 (0.5)*	6.2 (0.7)†	9.0 (0.5)‡	26.71 ^a
BDI ²²	1.1 (1.2)*	7.4 (1.5)†	12.0 (1.1)†	22.27 ^a
STAI ²³				
State	27.2 (1.4)*	38.4 (1.8)†	39.6 (1.3)†	23.76 ^a
Trait ^c	27.1 (1.7)*	41.0 (2.1)†	47.9 (1.6)‡	42.68 ^a

Abbreviations: BDI, Beck Depression Inventory; BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); BSQ, Body Shape Questionnaire; EDE, Eating Disorder Examination; NA, not applicable; STAI, State-Trait Anxiety Inventory; TFEQ, Three-Factor Eating Questionnaire.

*, †, ‡: Symbols that differ represent differences of $P < .05$ between groups after Bonferroni correction.

^a $P < .001$.

^b Analyses were completed comparing only bulimia nervosa and purging disorder groups because controls could have no symptoms.

^c $df = 2,82$ because 5 participants did not complete the STAI Trait Scale because they overlooked it.

Blood samples were collected into chilled sodium heparinized tubes and immediately centrifuged, and plasma was passed through C18 Sep-Paks (Waters Corporation, Milford, Massachusetts) to extract CCK, as previously described.^{11,25} Sep-Paks were stored at -70°C and shipped on dry ice by overnight mail to 1 of us (R.A.L.) for bioassay.²⁵ This procedure has shown high specificity for CCK relative to gastrin and high sensitivity to fasting CCK concentrations.²⁵ The bioassay detection limit was 0.2 pmol/L, and intraassay and interassay coefficients of variation were 7.4% and 10.4%, respectively.²⁵ Samples were analyzed in batches, with laboratory personnel blind to study group.

MEASURES

Clinical Interviews

The EDE¹⁸ was administered at study visit 1 to confirm diagnostic groups and to assess severity of eating abnormalities during the past 3 months. The EDE includes standardized questions to distinguish between objectively large binge episodes (OBEs) that are consistent with the DSM-IV definition of binge eating and episodes in which a person experiences a loss of control but has not eaten more than most people would eat under similar circumstances (subjective binge episodes [SBEs]). Based on data indicating that individuals without eating disorders will consume up to but not more than 1000 kcal,^{26,27} SBEs were defined as including less than 1000 kcal. In contrast, OBEs were defined as including at least 1500 kcal to minimize diagnostic misclassification. Thus, individuals whose typical or largest binge size fell

between 1000 and 1500 kcal were not eligible for participation. Three sources were used to calculate caloric content of binges: Life Form software,²⁸ online product information for commercially produced foods, and *Bowe's & Church's Food Values of Portions Commonly Used*.²⁹ Consistent with DSM-IV criteria, all binge episodes were required to occur within a 2-hour period. There was a significant difference in self-reported binge size between participants with BN (mean [SE], 2920 [335] kcal) and participants with PD (mean [SE], 750 [43] kcal) ($t_{37,22} = 6.42$; $P < .001$). For BN, binge size represents either typical or average OBE size over the previous 4 weeks. Participants with PD were asked to describe their largest binge for the previous 12 weeks. Thus, the estimated SBE size may be larger than typical for PD. Further, 2 participants with PD (10%) reported no binge eating. Reliability for eating disorder diagnoses was $\kappa = 1.0$ in the current study based on 15% of interviews randomly selected for reliability assessment. Interrater (Pearson r) and internal (Cronbach α) reliabilities for the EDE subscales were as follows: restraint, 1.0 and 0.87; eating concern, 0.998 and 0.81; weight concern, 0.995 and 0.90; and shape concern, 0.999 and 0.92.

The SCID-I¹⁹ was administered at study visit 1 to assess mood disorders ($\kappa = 0.65$), substance use disorders ($\kappa = 1.0$), anxiety disorders ($\kappa = 0.78$), and lifetime diagnoses of eating disorders. Reliabilities for lifetime eating disorder diagnoses were $\kappa = 0.65$ for AN and $\kappa = 1.00$ for BN.

Self-report Measures

The Beck Depression Inventory²² was completed at both study visits; values from study visit 2 are reported in Table 1. In the

Table 2. CCK Response to the Test Meal^a

Parameter	γ (SE)			
	A	B.1	B.2	C
Fixed effects				
Intercept	18.56 (0.63) ^b	15.99 (0.63) _b	14.00 (0.57) ^b	16.02 (1.65) ^b
Time		0.17 (0.04) ^b	0.97 (0.10) ^b	0.76 (0.14) ^b
Time squared			-0.03 (0.003) ^b	-0.03 (0.003) ^b _c
Assay				Control: 0.17 (0.06) ^d
Group × time				PD: 0.16 (0.07) ^e
				BN: 0 (0) ^f _c
Assay × time				
Random effects				
Intercept (time)	18.66 (5.16) ^b	0.04 (0.01) ^d	0.05 (0.01) ^b	0.02 (0.008) ^d
Deviance	1526.64	1516.98	1457.45	1384.41
Improvement in model fit		$\chi^2_1 = 9.66^d$	$\chi^2_1 = 59.53^b$	$\chi^2_{31} = 73.04^b$

Abbreviations: BN, bulimia nervosa; CCK, cholecystokinin; PD, purging disorder.

^aColumn "A" refers to unconditional means model; "B.1," unconditional growth model with time as a linear effect; "B.2," unconditional growth model with time and time squared for linear and nonlinear effects of time; and "C," final model. The dependent variable in these analyses is the square root of CCK values (to correct for positive skew) × 10 (to avoid problems with boundary constraints).³⁰ Effects of time are expressed in minutes.

^b $P < .001$.

^cParameter estimates (SE) for each level of assay and assay × time are available on request.

^d $P < .01$.

^e $P < .05$.

^fThe model sets this parameter to 0 to avoid redundancy.

current sample, internal consistency and test-retest reliability were 0.94 and 0.81, respectively. The Body Shape Questionnaire²¹ was administered at study visit 1. Internal consistency was 0.99. The State-Trait Anxiety Inventory²³ was completed at both study visits (study visit 2 values are reported in Table 1). Consistent with the construct of trait vs state anxiety, test-retest reliability was higher for the Trait (0.91) than the State Scale (0.73), whereas internal consistency was high for both the Trait (0.96) and State (0.95) scales in the current study. The Three-Factor Eating Questionnaire²⁰ was administered at study visit 1. Internal consistency was 0.94 for the Cognitive Restraint Scale, 0.92 for the Disinhibition Scale, and 0.85 for the Hunger Scale in the current study.

DATA ANALYSES

Clinical data collected at study visits 1 and 2 were analyzed using multivariate analyses of variance. Repeated measures collected at study visit 2 were analyzed using multilevel model analyses to examine within-subject change over time (level 1) and between-subject predictors of change over time (level 2). This approach was selected over repeated-measures analyses of variance because of our unequal group sizes and unbalanced design with regard to distribution of groups across assays. This approach allowed us to include assay and collection site (GCRC) as covariates in analyses of CCK response. One of us (R.A.L.), who remained blind to study group, reviewed bioassay results; only plasma CCK values falling on the linear portion of the standard curve were included in analyses of CCK response (n=76 participants, n=23 controls [a larger proportion of controls were excluded because of their overrepresentation in an assay that had limitations in the standard curve], n=19 participants with PD, n=34 participants with BN). Data were examined and corrected for skew and outliers, with transformations and sample sizes noted in the "Results" section. Bonferroni-corrected *P* values were used for post hoc comparisons.

RESULTS

CLINICAL COMPARISONS

Table 1 presents comparisons on clinical variables assessed at study visits 1 and 2. Both eating disorder groups scored significantly higher on all measures of eating abnormalities compared with non-eating disorder controls. No significant differences were observed between PD and BN on global eating disorder severity, purging frequency, dietary restraint, or body image disturbance. However, consistent with previous findings,¹ women with PD reported significantly lower disinhibition and hunger compared with women with BN. Both eating disorder groups reported higher levels of depression and anxiety compared with controls, and participants with BN reported higher trait anxiety compared with participants with PD. In contrast, participants with BN and PD did not differ significantly on depression or state anxiety. Although differences on depression approached traditional thresholds for significance ($P = .052$), this did not achieve significance with the Bonferroni-corrected threshold (Table 1). Similar results were obtained when restricting analyses to the 76 participants included in CCK analyses.

TEST-MEAL RESPONSE

CCK Response

Following recommendations by Singer and Willett,³⁰ we initially fit an unconditional means model that supported significant within-person variance in CCK levels (Table 2, model A). We then fit an unconditional growth

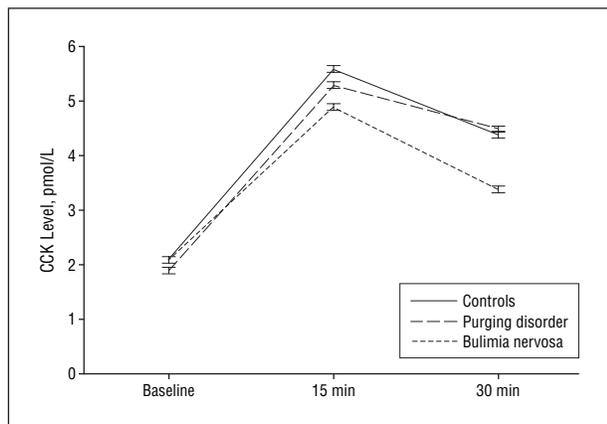


Figure 2. Least-squares mean (SE) cholecystikinin (CCK) levels at baseline, 15 minutes, and 30 minutes by group. There were 76 participants: 23 controls, 19 participants with purging disorder, and 34 participants with bulimia nervosa. Standard errors of estimates were allowed to vary over time in models. The mean standard error of estimates across time is presented.

model that confirmed significant within-person increases in CCK levels following test-meal consumption ($F_{1,175.57}=19.11$; $P<.001$), and model fit was significantly improved by adding a nonlinear effect of time (Table 2, models B.1 and B.2).

Level 2 (between-subject) variables (group, assay, and GCRC) and their interactions with time and time squared were included in an initial multilevel model. Demographic variables such as age and BMI were not included because these did not differ significantly among groups and because we had no a priori hypotheses regarding the influence of these variables on CCK response. Predictors with parameter estimates that did not differ significantly from zero were sequentially removed, and remaining parameters were examined for possible removal. Full maximum-likelihood estimates were used so that fit could be compared between nested models.³⁰ The Hessian matrix was positive definite and conversion criteria were satisfied for all models. The final model included significant effects for intercept (indicating that baseline CCK values differed significantly from 0; $F_{1,151.62}=634.46$; $P<.001$), time (indicating an increase in CCK values following ingestion of the test meal; $F_{1,185.22}=119.76$; $P<.001$), time squared (indicating a nonlinear change in CCK values over time; $F_{1,152.44}=92.59$; $P<.001$), assay (indicating significant interassay variability in CCK levels; $F_{13,151.72}=3.28$; $P<.001$), and group \times time interaction (indicating significant differences in CCK response among groups; $F_{2,76.01}=4.72$; $P=.01$) (Table 2, model C).

As predicted, participants with PD had significantly greater CCK responses compared with participants with BN ($t_{76.44}=2.51$; $P=.01$) but did not differ significantly from controls ($t_{75.93}=0.03$; $P=.98$). The CCK response was blunted in participants with BN compared with controls ($t_{76.61}=-2.67$; $P=.009$) (Figure 2). The final model did not include significant effects for group (indicating no significant differences among groups in baseline/intercept CCK values), GCRC, or GCRC \times time (indicating no significant site differences in CCK values or CCK response). Assay \times time was not a significant covariate; however, its removal resulted in worse model fit.

Subjective Responses

Figure 3 presents changes in subjective responses to the test meal by group for VAS ratings of “satiated (satisfied),” “full,” “hungry,” “urge to binge,” and “urge to vomit.” **Table 3** presents type 3 tests of fixed effects. For “satiated,” multilevel model analyses supported significant effects for time, time squared, and group. Participants with BN reported significantly lower satiation throughout test-meal procedures compared with controls ($t_{252.69}=-2.51$; $P=.01$) and participants with PD ($t_{252.69}=-2.23$; $P=.03$), who did not differ significantly from each other. However, there was no significant group \times time interaction. Post hoc comparisons of groups at each time indicated no significant differences after Bonferroni correction.

For “full,” analyses supported significant effects for time, time squared, and group \times time. Participants with PD reported significantly greater increases in fullness following the test meal compared with controls ($t_{79.92}=2.56$; $P=.01$) and participants with BN ($t_{79.71}=3.20$; $P=.002$), who did not differ significantly from each other. Post hoc comparisons of groups at each time indicated that participants with PD reported significantly higher levels of fullness 30 minutes following the test meal compared with controls and participants with BN.

For “hungry,” analyses supported significant effects for time, time squared, and group. Participants with PD reported significantly lower hunger throughout test-meal procedures compared with controls ($t_{241.73}=-3.51$; $P=.001$) and participants with BN ($t_{241.73}=-1.98$; $P=.049$), who did not differ significantly from each other. However, there was no significant group \times time interaction. Post hoc comparisons of groups at each time indicated that participants with PD reported significantly lower hunger at each assessment compared with controls.

Given posited associations between CCK response and satiation as well as different results for analyses of “satiated,” “full,” and “hungry,” we examined associations between CCK levels and subjective test-meal response. The CCK levels were significantly and positively associated with “satiated” (γ [SE], 0.56 [0.21]; $t_{231.34}=2.65$; $P=.009$) but not “hungry” (γ [SE], -0.25 (0.19); $t_{246.07}=-1.34$; $P=.18$). The Hessian matrix was not positive definite for analyses of “full.” Thus, CCK levels appeared to demonstrate a specific association with subjective ratings of satiation.

Analyses of “urge to binge” supported a significant group effect. Throughout test-meal procedures, participants with BN reported a significantly greater desire to binge compared with controls ($t_{72}=6.01$; $P<.001$) and participants with PD ($t_{72}=4.48$; $P<.001$), who did not differ significantly from each other. For “urge to vomit,” analyses supported significant effects of time, time squared, group \times time, and group \times time squared. Participants with PD and BN reported significantly greater increases in desire to vomit compared with controls ($t_{215.46}=8.98$; $P<.001$, and $t_{215.46}=9.18$; $P<.001$, respectively) but did not differ significantly from one another. Thus, test-meal responses on urges to binge and vomit were consistent with groups’ clinical presentations.

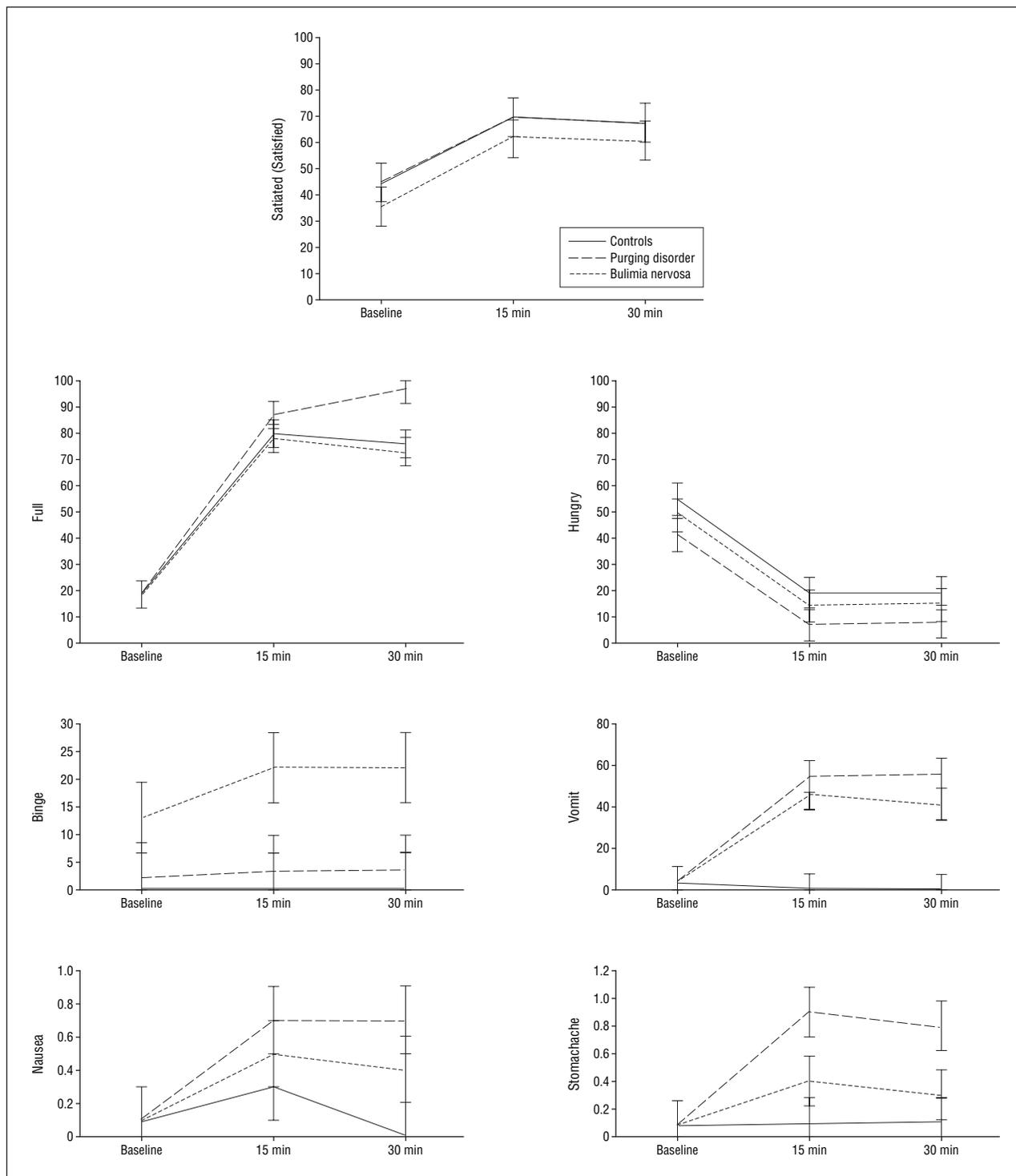


Figure 3. Subjective responses (least-squares mean [SE]) to test meal by group. Four participants (2 control participants and 2 participants with bulimia nervosa) did not complete visual analog scales because they overlooked booklets during study visit 2. Following exclusion of outliers, there were 76 to 86 participants: 25 to 28 controls, 32 to 35 participants with bulimia nervosa, and 15 to 20 participants with purging disorder across analyses of visual analog scale scores. Ninety participants were analyzed for the adverse effects of nausea and stomachache. Participants' standard errors of estimates were allowed to vary over time in models. The mean standard error of estimates across time is presented.

We conducted additional analyses of test-meal adverse effects related to symptoms of gastrointestinal distress, specifically nausea and stomachache. These ratings were made at baseline, 15 minutes, and 30 minutes postprandial with response options ranging from 0 (“not at all”) to 4 (“extremely”). For nausea, analyses sup-

ported significant effects of time, time squared, and group \times time. Compared with controls, participants with PD ($t_{89,94} = 2.72$; $P = .008$) and BN ($t_{89,94} = 2.43$; $P = .02$) reported significantly greater increases in nausea but did not differ significantly from each other. For stomachache, analyses supported significant effects of time, time

Table 3. Tests of Type 3 Effects for Subjective Test-Meal Responses

	Time	Time Squared	Group	Group × Time	Group × Time Squared
Satiated	$F_{1,194.73} = 43.31^a$	$F_{1,171.13} = 23.15^a$	$F_{2,252.69} = 4.00^b$	NS ^c	NS ^c
Full	$F_{1,181.17} = 550.89^a$	$F_{1,159.22} = 288.59^a$	NS ^c	$F_{2,79.88} = 5.36^d$	NS ^c
Hungry	$F_{1,191.39} = 93.25^a$	$F_{1,172.75} = 46.68^a$	$F_{2,241.73} = 6.19^d$	NS ^c	NS ^c
Urge to binge	NS ^c	NS ^c	$F_{2,72} = 21.26^a$	NS ^c	NS ^c
Urge to vomit	$F_{1,211.54} = 129.91^a$	$F_{1,143.04} = 85.53^a$	NS ^c	$F_{2,215.46} = 55.58^a$	$F_{2,143.58} = 33.00^a$
Nausea	$F_{1,220.25} = 23.11^a$	$F_{1,178.22} = 18.34^a$	NS ^c	$F_{2,89.94} = 4.63^b$	NS ^c
Stomachache	$F_{1,218.50} = 22.37^a$	$F_{1,177.21} = 13.41^a$	NS ^c	$F_{2,236.20} = 11.98^a$	$F_{2,177.44} = 6.24^a$
Sad	NS ^c	NS ^c	$F_{2,77} = 25.85^a$	NS ^c	NS ^c
Tense	NS ^c	NS ^c	$F_{2,82.54} = 14.25^a$	NS ^c	NS ^c

Abbreviation: NS, not significant.

^a $P < .001$.

^b $P < .03$.

^cNonsignificant effects were removed from the model; thus, no test values are available from the final model for nonsignificant effects.

^d $P < .01$.

squared, group × time, and group × time squared. Participants with PD reported significantly greater increases in stomachache compared with controls ($t_{236.20} = 4.89$; $P < .001$) and participants with BN ($t_{236.20} = 3.30$; $P = .001$), who did not differ significantly from each other.

Finally, we examined potential changes in VAS scores for “sad” and “tense” in response to the test meal. However, analyses indicated no significant effect of time. Instead, a significant group effect was found for “sad” and “tense.” Participants with BN reported greater sadness compared with controls ($t_{77} = 6.94$; $P < .001$) and participants with PD ($t_{77} = 4.30$; $P < .001$), who did not differ significantly from each other. Controls reported significantly lower levels of feeling tense compared with both participants with BN ($t_{82.59} = 4.29$; $P < .001$) and participants with PD ($t_{82.45} = 4.85$; $P < .001$), who did not differ significantly from each other. There were no significant group × time interaction effects for these measures.

Although there were no significant time or group × time effects for feeling tense, we conducted exploratory analyses to determine whether reports of greater postprandial nausea, fullness, or stomachache by participants with PD might be explained by tense feelings elicited by test-meal procedures. We entered feeling tense as a predictor along with other predictors and relevant interactions. Significant group × time interactions remained for fullness ($F_{2,80.50} = 4.30$; $P = .02$), nausea ($F_{2,199.10} = 3.30$; $P = .04$), and stomachache ($F_{2,196.69} = 5.12$; $P = .007$). Thus, greater postprandial fullness and gastrointestinal distress in PD do not appear to be attributable to increased feelings of tension during the test-meal session.

COMMENT

Participants with PD reported significant elevations on measures of eating abnormalities compared with controls, consistent with previous research.^{1,4} In addition, participants with PD did not differ significantly from participants with BN on indicators of eating disorder severity, consistent with some,^{1,2,31} but not all,^{3,4} previous stud-

ies. Taken together, results support the clinical severity of PD. We replicated an earlier finding of significantly lower scores on the disinhibition and hunger subscales of the Three-Factor Eating Questionnaire in PD compared with BN.¹ Assessment of CCK response to a standardized test meal supported hypotheses that participants with PD would have a significantly greater postprandial CCK response compared with participants with BN. In addition, participants with PD reported significantly greater satiation throughout the test meal and greater postprandial fullness compared with participants with BN. Participants with PD did not differ significantly from control participants on CCK response or satiation but did report significantly greater postprandial fullness. Of note, examination of fullness ratings revealed that participants with PD described themselves as “extremely full” (mean score of 97 on a 100-mm VAS) 30 minutes following the test meal compared with controls and participants with BN, whose fullness ratings had subsided to scores of approximately 75. Thus, both subjective and objective physiological responses to the standardized test meal are consistent with study hypotheses and the clinical presentation of PD.

We replicated blunted CCK response in participants with BN compared with controls, as has been reported in other studies.¹¹⁻¹³ Further, participants with BN reported lower satiation throughout test-meal procedures, and subjective ratings of satiation were associated with CCK levels. However, change in satiation following the test meal did not differ between groups, and participants with BN did not report significantly less postprandial fullness compared with controls. Our results may reflect the nature of the test meal used in the current investigation. We selected a test meal of 900 kcal in 600 g of fluid based on procedures used by Devlin et al.¹² Similar to the current investigation, Devlin et al¹² found significantly blunted postprandial CCK release in participants with BN compared with controls but did not find significant group differences in subjective ratings of fullness or hunger. To our knowledge, only 1 study¹¹ using a standardized test-meal procedure to assess CCK response has demonstrated significantly lower subjective

ratings of postingestive satiety in BN. Other studies either have not reported^{32,33} or have not found significant differences between groups,¹² and 1 study reported higher ratings of satiety in BN.¹³ In contrast, well-replicated findings of blunted satiety ratings in BN have come from feeding laboratory studies using an ad lib test-meal procedure in which participants with BN consume more food than control participants.^{15,34,35} In the present study, participants could not consume different amounts of food. Instead, all participants completed the standardized liquid test meal, and the caloric content of our test meal best approximated a subjective binge episode, not an objective binge episode.²⁷ Thus, our test meal may not have involved enough food to elicit different fullness ratings between controls and participants with BN. Future research using an ad lib test-meal procedure would be important for evaluating behavioral differences in food intake among groups as an objective measure of satiation.

Blunted CCK response has been posited as a possible cause, consequence, and maintenance factor for binge eating in BN.^{11,12,14} However, no previous study has sought to determine whether blunted CCK response is specific to the symptom of binge eating in BN. Instead, studies have compared CCK response in participants with BN and healthy control participants who differ on a number of features, including binge eating, self-induced vomiting, and dietary restraint. The current study significantly advances understanding of the association between binge eating and CCK function because participants with BN and PD only differed on the presence of binge eating and features linked to this symptom.

In addition to supporting a specific link between the symptom of binge eating and blunted CCK response, this study supports an objective physiological distinction between PD and BN. Further, results provide initial clues for why women with PD feel compelled to purge after consuming an amount of food that most people would regard as normal. Such amounts may be more likely to elicit feelings of extreme fullness, nausea, and stomachache in women with PD. Symptoms of gastrointestinal distress do not appear to be a consequence of increasing tension in response to the test meal. Instead, these symptoms may reflect other physiological responses to food intake, such as increased postprandial responses in other neuropeptide systems that regulate food intake³⁶ or delayed gastric emptying.³⁷ Research on psychobiological factors that contribute to the development or maintenance of binge eating vs purging is important for identifying new targets for therapeutic interventions for both PD and BN.

To our knowledge, the current study is the first to explore physiological as well as clinical features associated with PD. Strengths include careful clinical assessments using measures with strong psychometric properties, larger sample size and statistical power relative to previous studies of CCK response in BN,^{11-13,32,33} and use of statistical methods ideally suited to nonrandom, unbalanced designs. Despite its strengths, this study had certain weaknesses. First, we were unable to recruit an equal number of participants for the PD group compared with the BN and control groups. It is possible that study procedures may have biased recruitment of participants with PD to

include only those who were willing to consume the test meal. If so, results may underestimate differences in physiological and subjective postprandial responses in PD compared with BN. Further, although community-based recruitment reduces the potential impact of the Berkson bias, participants in our study may differ from patients encountered in clinical settings. Thus, care should be taken in generalizing results.

Second, we examined subjective and physiological responses to the test meal during the active phase of illness for our eating disorder groups. Thus, it is not possible to determine whether differences in CCK function represent a cause, consequence, or maintenance factor for binge eating in BN.

Third, assessments of subjective test-meal response were based on single-item VAS scales for which reliability and validity have not been clearly established. We selected this method because it has been used in previous test-meal studies. However, it is not clear that all participants understand what is meant by terms like "satiation," and attempts to clarify this by including "satisfied" may not capture this construct. Thus, more work is needed to create good measures for subjective responses to food intake.

Fourth, with the exception of increased fullness and stomachache in PD, other factors that differentiated PD from BN tended to indicate greater dysregulation in BN. Although these findings could be interpreted as demonstrating that BN is a more severe eating disorder compared with PD, we suspect that results reflect the extent to which more is known about factors that contribute to binge eating relative to factors that contribute to purging. Purging has most often been studied in the context of BN and most often viewed as a consequence of binge eating. Thus, little is known regarding factors that uniquely contribute to the propensity to purge among those who do not binge eat. Recent studies suggest that these individuals may outnumber those who purge in response to binge eating,^{5,38} emphasizing the need for more work on this topic.

Findings are important in further supporting the clinical significance of PD on measures of eating disorder severity as well as indicating its distinctiveness from BN on measures of subjective and physiological responses to a test meal. Results add to the growing literature suggesting that a diagnosis of PD should be considered for inclusion in the classification of eating disorders. Based on the adage that "we study what we define,"³⁹ elevation of PD from the undifferentiated eating disorder not otherwise specified category would facilitate much needed research into the causes, consequences, and, most importantly, efficacious treatment of this serious eating disorder.

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