Published in final edited form as:

Physiol Behav. 2008 February 27; 93(3): 481–485.

Effect of eating rate on binge size in Bulimia Nervosa

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Abstract

Effect of eating rate on binge size in bulimia nervosa. Bulimia Nervosa (BN) is an eating disorder characterized by recurrent episodes of binge eating. During binge eating episodes, patients often describe the rapid consumption of food, and laboratory studies have shown that during binges patients with BN eat faster than normal controls (NC), but the hypothesis that a rapid rate of eating contributes to the excessive intake of binge meals has not yet been experimentally tested. The aim of this study was to assess the effect of eating rate on binge size in BN, in order to determine whether binge size is mediated, in part, by rate of eating. Thirteen BN and 14 NC subjects were asked to binge eat a yogurt shake that was served at a fast rate (140g/min) on one occasion and at a slow rate (70g/min) on another. NC subjects consumed 169 g more when eating at the fast rate than when eating at the slow rate. In contrast, consumption rates failed to influence binge size in patients with BN (fast: 1205 g; slow: 1195 g). Consequently, there was a significant group by rate interaction. As expected, patients with BN consumed more overall than NC subjects (1200 g vs. 740 g). When instructed to binge in the eating laboratory, patients with BN ate equally large amounts of food at a slow rate as at a fast rate. NC subjects ate less at a slow rate. These findings indicate that in a structured laboratory meal paradigm binge size is not affected by rate of eating.

Keywords

bulimia nervosa; eating laboratory; eating rate; binge eating; eating disorders

1. Introduction

Bulimia Nervosa (BN) is a psychiatric illness characterized by recurrent episodes of binge eating which are followed by inappropriate compensatory behaviors such as self-induced vomiting or laxative abuse. By definition, individuals with BN consume large amounts of food during their binge meals, and patients typically report that they eat rapidly during binges [1]. Both large meal size and rapid consumption have been documented in laboratory studies of binge eating in BN [2-4]. The centrality of rapid eating in BN, in addition to receiving support from clinical description and laboratory studies, has been proposed as a possible target for intervention [5]. In addition, slowing eating is prominently featured as a method for reducing meal size in popular behavioral weight control programs for obese individuals [6]. However, the empirical basis for this treatment approach has not yet been established.

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The aim of this experiment was to test the hypothesis that binge meal size of patients with BN is strongly influenced by rate of eating. We hypothesized that, during a binge meal, both patients and controls would consume more when eating rapidly than when eating slowly, and that this difference would be greater in patients than in controls.

2. Methods and Materials

2.1 Subjects

Nineteen adult women meeting DSM-IV criteria for BN were recruited to participate in this study. These patients were seeking in- or out-patient treatment for their eating disorder, and were between the ages of 18 and 45 years and between 80 and 120 percent of ideal body weight for their height [7]. Patients used vomiting as one of their primary methods of compensating for binge eating, and were currently free of psychotic illness, drug abuse, and significant suicidal ideation. Patients were required to have ceased taking any medication other than oral contraceptives for at least two weeks (for at least six weeks for fluoxetine), and were studied before the initiation of treatment for their eating disorder.

Nineteen women without a history of eating disorders or of significant psychiatric disorders, meeting the same age and weight criteria, were recruited through advertisements as normal control subjects and received monetary compensation in return for their participation. Control subjects were not attempting to lose weight and were taking no medications other than oral contraceptives. Both patients and controls were in good general physical health. This study was reviewed and approved by the Institutional Review Boards of the New York State Psychiatric Institute and St. Luke's Roosevelt Hospital Center. Written informed consent was obtained from all subjects.

2.2 Apparatus

A specialized apparatus was constructed for this study. A clear plastic box was placed in front of subjects, behind which was a box containing a pump and food reservoir cooled by an icepack. The clear box held a cup containing the strawberry yogurt shake meal. The cup was marked with two black lines, between which the subjects were asked to keep the level of shake which was pumped into the cup by remote control. A straw extended from the cup to the outside of the box from which the subject consumed the shake. This strawberry yogurt shake is palatable and has been used successfully in our previous laboratory studies of binge eating in BN [4,8]. The staff set the rate for delivery and observed subjects on closed circuit TV to ensure directions were followed. By eating fast or slow enough to keep the level of the shake between the two black lines, subjects effectively ate at the same speed at which the cup was being filled.

2.3 Daily Procedure

All subjects visited the laboratory for two test days that followed an initial screening visit. After an overnight fast, subjects arrived at the laboratory at 0900 for a standardized 300 kcal breakfast that consisted of one Thomas' English muffin with 1½ pats of butter, and 250 g of Red Cheek Natural apple juice. Subjects were free to leave the laboratory and were instructed to return 5½ h later, without eating or drinking anything other than water in the interim. On the screen day subjects completed demographic and health-related forms, and were then given a taste test to determine whether they liked the test meal. Only participants who rated the shake at least a '6' (= like slightly) on a 9-point category scale of liking [9] were accepted. After the taste test, subjects were asked to eat the strawberry yogurt shake until the researcher returned to the eating room. The meal was delivered at a rate of 70 g/min until 225 g had been consumed, at which point the meal was delivered at 140 g/min. The meal was terminated once the subject had consumed 450 g. Those subjects who were able to follow the procedures returned for the two subsequent test trials, which took place on non-consecutive days.

Subsequent test trials also began with the 300 kcal breakfast after an overnight fast. Six hours later, subjects were served the strawberry yogurt shake and were instructed to “let yourself go and binge eat.” On one of the two days the yogurt shake was delivered at the slow rate, and on the other day the shake was delivered at the fast rate. The order of presentation of the two rates was counterbalanced across subjects. Subjects were asked to report whether they binged and vomited after each meal. To be included in the data analysis, subjects had to report that they liked the test meal, were able to follow instructions, and patients with BN had to have binged on at least one of the test meals.

2.4 Experimental Design and Data Analysis

This study design was a two-way, mixed-model design, with group (BN and control) as the between-subjects factor and rate of eating (fast and slow) as the within-subject factor. The order of the presentation of delivery rates was counterbalanced. The dependent variable was the amount consumed in the test meal. Statistical analysis was performed using the SAS Proc Mixed procedure.

Data from 13 patients with BN and 14 normal control subjects were used in the analysis. Of the nineteen normal control participants who began the study, two did not continue after the screen/adaptation meal because they did not like the shake meal; one subject moved away after beginning the protocol; two normal control subjects were excluded from the data analysis because post hoc inspection of the data indicated that the shake was delivered to the container at the slow rate on both study days. Of the nineteen patients with BN who began the study, one patient withdrew from the study after the screen/adaptation meal because she did not feel that she could comply with the instructions to binge eat; one patient was dropped after the screen/adaptation meal because she found the shake meal too rich; one patient did not show up for her second test meal; one patient with BN was excluded from the data analysis because she consumed all of the experimental meal, which did not allow us to measure the full difference in *ad libitum* consumption at the two eating rates. Two subjects with BN were excluded from the data analysis because they reported that they did not binge eat during the procedure.

3. Results

The mean age was 26.31 ± 1.42 y for patients with BN and 25.07 ± 0.94 y for normal controls. Mean BMI was 21.37 ± 0.46 kg/m² for patients with BN, and 21.54 ± 0.44 kg/m² for controls. Patients had been ill for an average of 9.92 ± 1.66 y and binged and vomited an average of 10.13 ± 2.17 times per week.

The manipulation of the rate of consumption was successful, and all the subjects ate substantially faster during the fast condition than the slow. Although there was no difference in the rate of consumption between the two groups in the slow condition (72.3 ± 5.2 g/min for BN, 72.1 ± 5.1 g/min for NC), the rate of consumption in the fast condition for the BN group was greater than that in the control group (167.8 ± 5.2 g/min for BN, 146.1 ± 5.1 g/min for NC). Despite the greater difference in eating rates in the fast and slow conditions among the patients with BN, there was no effect of eating rate on amount consumed in these subjects (see results below).

The mean meal sizes for the two groups are shown in Table 1. Although meal size was significantly larger for the fast rate compared to the slow rate, there was a significant rate $\times$ group interaction. For the control group the mean difference in consumption between fast and slow rates was 168.9 grams ± 53.16 SE, whereas for patients it was only 10.79 grams ± 54.77 SE. Patients with BN ate significantly more than normal controls at both rates.

Although there was no significant main effect of order of meals, i.e. slow-fast vs. fast-slow sequence ($F(1,23)=0.079, p<0.78$), there was a significant rate $\times$ order interaction (a trial effect) ($F(1,23)=5.48, p<0.05$). Subjects assigned the slow-fast sequence consumed 179.1 ± 54.8 g more in the fast condition, while there was no difference in intakes between the fast and slow conditions in subjects assigned to the fast-slow sequence (i.e. the difference was 0.45 ± 53.2 g). There was no significant group $\times$ order interaction ($F(1,23)=0.20, p=0.66$). There was no significant group $\times$ rate $\times$ order interaction ($F(1,23)=0.19, p=0.67$), but the tendencies for both BN and NC subjects to eat more in the second meal, and for control subjects but not subjects with BN to eat more in their fast meal yielded group specific rate/order patterns (Table 2). Although it was not significant, patients with BN assigned to the slow-fast sequence consumed 116.7 ± 74.4 g more in the second meal, and patients assigned to the fast-slow sequence consumed 95.1 ± 80.4 g less in the second meal. In contrast, normal control subjects assigned to the slow fast sequence ate 241.4 ± 80.4 g more in the second meal and controls assigned to the fast-slow sequence consumed 96.0 ± 69.6 g more in the second meal.

4. Discussion

The goal of this experiment was to measure the effect of rate of eating on the size of a binge meal among patients with BN and controls. We were able to manipulate rate of eating in the laboratory successfully in both groups. The principal finding of the study was that ingestion rate affected binge size only in the normal control subjects, not in subjects with BN. The patients with BN did not need to eat quickly in order to binge, and reducing their rate of eating failed to influence binge size. However, manipulating rates of consumption did affect meal size for control subjects. Both the demonstration of an effect of rate of eating on meal size in controls and the lack of such an effect in patients with BN are of interest.

Although several previous feeding laboratory studies have demonstrated that obese individuals eat more rapidly than normal weight individuals [10], previous studies have not directly manipulated eating rate. The amount of food eaten in a meal depends on a number of changes that occur over time following the beginning of the meal, including gastric distension, passage of food into the small intestine, and the subsequent release of various satiety-mediating peptides. In theory, more rapid eating may allow the consumption of a greater amount of food prior to the development of physiological inhibitory signals that contribute to termination of the meal and thus determine its size. The current study did not measure any of these physiological signals; thus, we were unable to determine the relationship between food ingestion and the time course of post-ingestive signals related to meal termination. However, there is considerable evidence that controls of eating that operate in normal subjects are attenuated or delayed in individuals with BN, possibly rendering them less responsive to the signals that would limit eating at a slower rate [11] [12] [13].

The lack of an effect of rate of consumption on meal size in patients with BN, in contrast with the observed effect in normal controls, suggests that the large size of binge meals in BN is not attributable simply to a rapid eating rate during a binge meal. Slowing the rate of eating of individuals with BN during a binge meal did not significantly decrease meal size. Conversely, increasing the rate of consumption of normal controls, while it does increase the amount consumed, did not immediately lead to the consumption of meals of comparable size to those of patients with BN. Taken together, these findings suggest that the signals, or responses to the signals, that lead to meal termination in a binge meal in BN patients may differ from those in normal subjects. For example there is evidence that the release of CCK, an important satiety signal, is diminished in patients with BN [11,14]. It is additionally possible that individuals with BN continue eating after meal-terminating signals reach values that terminate meals in controls, or that they are eating to achieve a different end point, such as the ability to readily induce vomiting. In fact, in a single case study, Azrin et al. [15] found that the instruction to

eat at a faster rate was associated with increased desire to vomit in a patient with BN. In our study, subjects were queried in a postmeal checklist about several sensations they may have had during the meal, including whether they felt like vomiting. All of our patients with BN reported feeling like vomiting, and did vomit after meals delivered at slow and fast rates, while none of the controls reported feeling like vomiting or vomiting.

It is important to note that the meal instruction, while the same for both groups, may have had a different meaning for the two groups. By definition, patients with BN had experience with recurrent binge eating while controls did not. Therefore, in the BN group, the instruction to “binge eat” may have prompted a well-practiced behavior that was not sensitive to manipulation of rate of eating. It is possible that in other circumstances, for example, in the absence of an instruction to binge eat, manipulation of rate of eating would have a greater impact on amount consumed in individuals with BN.

An unexpected finding of the study was that, despite the use of an adaptation meal, subjects overall ate 89.5 g more in the second than the first laboratory meal, giving rise to a rate by order interaction. The trial effect (rate $\times$ order interaction) was not significantly different in the patients with BN and controls. In subjects assigned to the slow-fast sequence this order effect augmented the effect of the rate instruction, while in subjects assigned to the fast-slow sequence, the rate and order effects negated each other. While our prior studies [16] have demonstrated that laboratory meal intake stabilizes after an adaptation meal, we did not instruct subjects to binge eat in those studies. We do not know how many adaptation meals are required for laboratory binge meal intake to stabilize, and it may be well for future investigations to note this caveat.

Some caveats are in order. Although laboratory studies presumably reflect the same physiological controls that would operate in any environment, they may not replicate all the controls of eating in an individual's usual environment, particularly cognitive and social controls. Our study was also limited to the consumption of a single food given at two predetermined rates accompanied by the instruction to binge eat. Whether similar results would be observed under other experimental conditions, such as faster or slower rates, with solid or even other liquid foods, without an explicit instruction to binge eat, or with manipulation of participants' emotional states via mood induction is unknown. Finally, since subjects were not instructed to eat either faster or slower, but simply to eat to keep the level in the cup constant, these results do not directly address the clinical impression that telling patients to eat more slowly helps them reduce intake.

A series of studies by Spiegel [17] examining the effect of bite size and ingestion rate on food intake on lean and obese subjects, found that ingestion rate did not determine meal size in lean or obese groups. While the current study also failed to demonstrate a relationship between experimentally controlled eating rate and binge meal size in patients with BN, our study did find an effect of eating rate on intake in control subjects. Rapid eating remains a salient feature of binge eating in BN, but this study suggests rapid eating may not be an essential element of binge eating in BN. Our results do not, however, imply that rate of eating plays no role in binge eating, nor that interventions focusing on modifying the rate of eating would necessarily be ineffective. Further laboratory studies using different instruction, e.g. an instruction to eat rapidly or slowly, rather than to binge eat or refrain from binge eating, may further elucidate the relationship among rate of eating, meal size and desire to vomit in individuals with eating disorders.

Acknowledgements

This work was supported by NIMH grant MH-42206 and Obesity Core Center NIH Grant DK-26687. A preliminary report of this work has appeared: JL Guss, HR Kissileff, M Torres, H Lofink, MJ Devlin, E Zimmerli, BT Walsh. Effect of eating rate on binge size in bulimia nervosa (BN). *Appetite* 44:352; 2005, abstract.

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Table 1Test meal data for patients with Bulimia Nervosa and Normal Controls in Fast and Slow conditions¹

Bulimia Nervosa (N=13)	Intake (g)
Fast	1205.75 ± 109.67 ²
Slow	1194.96 ± 109.67
Normal Controls (N=14)	
Fast	823.99 ± 106.46
Slow	655.28 ± 106.46

¹ All values are Mean ± SE.² A mixed model ANOVA using group as a between subjects factor and rate condition (fast or slow) as a within subjects factor found a significant main effect of group ($F(1,23) = 9.69$, $p < 0.01$) and rate instruction ($F(1,23) = 5.53$, $p < 0.05$) as well as a significant group by rate interaction ($F(1,23) = 4.28$, $p < 0.05$). See text for means and SEs of differences. Note: means are unweighted and error terms are pooled estimates generated by maximum likelihood estimators in SAS Proc Mixed.

Table 2

Test meal intake (g) for patients with Bulimia Nervosa and Normal Controls assigned to Slow-Fast and Fast-Slow sequence of meals¹

	Slow-Fast Sequence (N=7)	Fast-Slow Sequence (N=6)
Bulimia Nervosa (N=13)		
Fast	1204.7 ± 149.0 ²	1206.8 ± 160.9
Slow	1088.0 ± 149.0	1301.9 ± 160.9
Fast -Slow Diff	116.7 ± 74.4	-95.1 ± 80.4
Normal Controls (N=14)	(N=6)	(N=8)
Fast	872.7 ± 160.9	775.3 ± 139.4
Slow	631.3 ± 160.9	679.3 ± 139.4
Fast -Slow Diff	241.4 ± 80.4	96.0 ± 69.6

¹ All values are Mean ± SE.

² A mixed model ANOVA using group as a between subjects factor and rate condition (fast or slow) and order of meals (slow-fast or fast slow) as within subjects factors, found a significant rate × order interaction ($F(1,23)=5.48$, $p<0.05$). There was no significant main effect of order ($F(1,23) = 0.079$, $p=0.78$), no significant group × order interaction ($F(1,23) = 0.20$, $p=0.66$) and no significant group × rate × order interaction ($F(1,23) = 0.19$, $p=0.67$). Note: means are unweighted and error terms are pooled estimates generated by maximum likelihood estimators in SAS Proc Mixed.