Published in final edited form as:
Int J Eat Disord. 2009 March ; 42(2): 173–178. doi:10.1002/eat.20600.

Maintenance Factors for Persistence of Bulimic Pathology: A Prospective Natural History Study

Cara Bohon, M.S.[1], **Eric Stice, Ph.D.**[2,3], and **Emily Burton, Ph.D.**[3]

[1] Department of Psychology, University of Oregon, Eugene, Oregon

[2] Oregon Research Institute, Eugene, Oregon

[3] Department of Psychology, University of Texas at Austin, Austin, Texas

Abstract

Objective—To characterize the natural course of bulimia nervosa and identify potential maintenance factors that predict persistence of bulimic pathology in order to advance knowledge of processes that perpetuate this eating disturbance and permit the design of more efficacious treatments.

Method—We followed 96 women with threshold or subthreshold bulimia nervosa over a 1-year period with quarterly interviews.

Results—There were high rates of remission and relapse on a month-to-month basis, but remission became more likely to persist after a period of approximately 4 months of symptom abstinence. Initial elevations in thin ideal internalization, expectations for reward from eating, and binge frequency predicted greater time to remission of binge eating. Initial elevations in dietary restraint and compensatory behavior frequency predicted greater time to remission of compensatory behaviors.

Conclusion—Results imply that treatments for eating disorder may be more effective if they can reduce thin-ideal internalization, eating expectancies, and ineffective dieting, and produce rapid cessation of binge eating and compensatory behaviors.

Keywords

bulimia nervosa; maintenance factors; thin-ideal internalization; dietary restraint

Threshold and subthreshold bulimia nervosa is prevalent and marked by functional impairment and comorbidity [1]. Bulimic pathology is often chronic: a community-recruited natural history study found that only 49% of women with bulimia nervosa showed lasting remission from bulimic behaviors over 5 years [2], which is similar to the recovery rate that occurred over a 5-year follow-up of women treated with cognitive behavioral therapy (CBT [3]). Several long-term studies of treatment-seeking samples have found that the course of bulimic pathology is characterized by relapse and diagnostic crossover and have identified baseline factors that predict lasting recovery (e.g., [4–6]). Greater body image disturbance and worse psychosocial functioning predicted relapse in a sample of women seeking treatment for an eating disorder [5]. However, the fact that 55–75% of individuals with bulimic pathology do not seek treatment and a substantial number of those who do seek treatment have subthreshold bulimia nervosa (and are often excluded from treatment trials) suggests it would be valuable to examine the course and predictors of course in non-

Address correspondence to Cara Bohon at Department of Psychology, 1227 University of Oregon, Eugene, OR 97403.

treatment seeking samples [1,7]. It is vital to identify maintenance factors that predict persistence of bulimic pathology because it would inform maintenance theories of this disorder and may permit the design of more efficacious treatments. The fact that the current treatment of choice for bulimic pathology (CBT) only results in lasting remission for about 40% of those that begin treatment [1] suggests it is crucial to identify new maintenance factors so that could be targeted in treatment to improve recovery rates.

Only two prospective studies have investigated predictors of bulimic symptom persistence using non-treatment samples. Fairburn et al. [8] conducted diagnostic interviews with 102 women with threshold bulimia nervosa recruited from the community every 18 months over a 5-year period; elevated binge frequency, overvaluation of weight and shape, social maladjustment, a history of childhood obesity, and persistent compensatory behaviors predicted persistence of binge eating, whereas only persistence of binge eating predicted persistence of compensatory behaviors. Stice and Agras [9] administered a survey assessing bulimic symptoms at baseline and at 9-month follow-up among adolescent girls from high schools: elevated thin-ideal internalization at baseline predicted persistent binge eating and elevated body dissatisfaction and dietary restraint at baseline predicted persistence of compensatory behaviors. Although these two studies have advanced our understanding of maintenance factors, given that (a) only one studied individuals with clinically significant bulimic pathology, which were assessed with diagnostic interviews, (b) neither enrolled individuals with subthreshold bulimia nervosa, even though these individuals often present for treatment, (c) neither involved frequent diagnostic interviews (introducing potential recall bias), and (d) each examined a limited set of putative maintenance factors, it seemed prudent to conduct a new prospective study that attempted to address these issues. It is particularly vital to study individuals with subthreshold bulimia nervosa because this is a common presenting problem that is associated with marked subjective distress and functional impairment [6,10–11].

Accordingly, the goal of the present study was to investigate maintenance factors that predict persistence of bulimic pathology in a non-treatment sample of individuals with threshold or subthreshold bulimia nervosa using frequent diagnostic assessments. We recruited 96 women with bulimic pathology and followed them over a 1-year period with structured interviews every 3 months. Because elevated hunger, depressive symptoms, and the belief that eating improves mood theoretically increase the likelihood of binge eating and because dietary restraint and frequent use of use of unhealthy compensatory behaviors would reduce weight gain secondary to binge eating, we hypothesized that each might serve as a maintenance factor for binge eating. Because elevated thin-ideal internalization, body dissatisfaction, and binge eating frequency would theoretically increase the drive to use radical and unhealthy weight control behaviors, we hypothesized that each might serve as a maintenance factor for compensatory behaviors. As several of these variables (e.g., depressive symptoms, affect regulation eating expectancies, and hunger) have not been investigated in prior maintenance factor studies, the present study may identify new targets for treatment interventions. We also examined time to recovery and subsequent relapse to characterize the course of this disorder in a non-treatment sample. Further, the inclusion of participants with subthreshold bulimic pathology in a non-treatment sample allowed us to investigate similarities and differences in the chronicity of subthreshold and threshold levels of bulimia nervosa.

Method

Participants and Procedures

Participants were 96 young women (*M* age = 19.7 [SD=4.8]) who met threshold (*n*=42) and subthreshold (*n*=54) criteria for bulimia nervosa (11% Asian, 4% Black, 13% Hispanic, 1%

Native American, and 66% White). Participants were recruited from a large university and surrounding community with ads inviting women to take part in a longitudinal study of the course of eating disturbances (63 were college students [66%]). Of the 130 women responding to the ads, 101 were currently engaging in bulimic pathology, and 96 enrolled in the study. After providing informed consent, they completed a semi-structured diagnostic interview assessing eating disorder symptoms at baseline and every three months for the following year and a baseline survey that measured potential maintenance factors. They were compensated $10 for completing each assessment. The local institutional review board approved the study. Data were collected from 2000–2002.

Maintenance Factors

The Beliefs About Appearance Scale, which measures the belief that achieving the thin-ideal improves relationships, achievement, self-view, and mood, assessed thin-ideal internalization: this 20-item scale has shown internal consistency (α = .95), 3-week temporal reliability (*r* = .83), and convergent validity [12]. Items were averaged, as were items for the scales described subsequently. A 9-item Body Dissatisfaction measure assessed satisfaction with various body parts (e.g., waist, thighs) and has shown internal consistency (α = .94), 3-week test-retest reliability (*r* = .90), and predictive validity for future onset of bulimic symptoms [11]. The Restraint scale from the Three Factor Eating Questionnaire (TFEQ) assessed attempted dietary restraint; it has shown internal consistency (α = .63–.67) and 1-year test-retest reliability (*r* = .81; [13]). The Beck Depression Inventory assessed the intensity of depressive symptoms; it has shown internal consistency (α = .73–.95), test-retest reliability (*r* = .60–.90), and convergent validity with clinician ratings of depressive symptoms (*M r* = .75; [14]). The TFEQ-Hunger Scale assessed hunger; it has shown internal consistency (α = .63–.73) and 1-year test-retest reliability (*r* = .75; [13]). The Eating is Pleasurable and Useful as a Reward and the Eating Helps Manage Negative Affect subscales from the Eating Expectancies Inventory were used to assess eating expectancies; it has shown internal consistency (α = .90), criterion validity with eating disorder symptoms, and predictive validity for bulimic symptom onset [15].

Bulimic symptoms

The diagnostic items from the Eating Disorder Diagnostic Interview [11], a semi-structured interview, assessed DSM-IV criteria for bulimia nervosa over the past 3-months at each assessment. Participants met threshold criteria for bulimia nervosa if they engaged in binge eating and compensatory behaviors an average of twice a week for at least 3 months and reported that weight/shape was “definitely one main aspect of self-evaluation”. Following Crow et al., [10], to receive a diagnosis of subthreshold bulimia nervosa participants had to report engaging in binge eating and compensatory behaviors at least weekly for at least 3 months and report that weight/shape was “definitely an aspect of self-evaluation”. Such subthreshold cases would warrant a diagnosis of ED-NOS [16]. The symptom composite from this interview has shown internal consistency (α = .96), 1-month test-retest reliability (r = .95), convergent validity with alternative measures of eating pathology, and sensitivity to detecting intervention effects; the eating disorder diagnoses from this interview have shown 1-week test-retest reliability (κ = .96) and inter-rater agreement (κ = .86) in previous studies (e.g., [11]). Remission was defined as being symptom-free for at least 4 consecutive months, as this permitted adequate sensitivity to predicting remission (about 30% of the sample showed remission).[1] Relapse is defined as engaging in two or more episodes of binge eating or compensatory behavior after a period of remission.

Statistical Analyses

In order to explore the natural course of bulimia nervosa in the sample, we conducted descriptive analyses to characterize frequency of remission and mean time to remission. To

determine whether frequency of remission differed between those with subthreshold or threshold level symptoms, we conducted chi-square tests. In order to predict time to remission, we first conducted proportional hazards regression models to test the predictive effects of each potential maintenance factor. Predictors were standardized to make it easier to directly compare hazard ratios (HR), which were inverted so that scores above 1.0 reflect greater likelihood of experiencing symptom persistence for each one-unit increase of the predictor. We estimated multivariate models with predictors that showed significant prediction in the bivariate models.

Results

Preliminary Analyses

Participants with missing data on any study variable (n=27) did not differ significantly from those with complete data on any of the other measures, except body satisfaction (those with missing data had lower body dissatisfaction t [94] = 9.41, $p < .001$). Attrition was low, with 93% providing at least some follow-up data throughout the study. At the 4 follow-up assessments, we had the following number of participants: 94, 93, 93, and 89. We used full information maximum likelihood estimation to impute missing data because this approach produces more accurate parameter estimates than list-wise deletion. Means, correlations, and standard deviations of all variables are included in Table 1.

Course of Bulimia Nervosa

There were high rates of symptom remission and relapse during the follow-up. We examined remission and relapse for binge eating and compensatory behaviors separately because the concordance between the two behavioral symptoms of this disorder was moderate (M κ = .16), as was the case in a prior natural history study of bulimia nervosa [8]. Of the 96 participants, 31 (32%) experienced a period of binge remission lasting at least 4 months and 27 (28%) experienced a period of compensatory behavior remission lasting at least 4 months during the 1-year follow-up. Of those showing remission lasting 4 months or longer, the average time to remission was 2.7 (SD = 2.1) and 3.3 (SD = 2.8) months for binge eating and compensatory behaviors respectively. Symptom remission was more likely for subthreshold cases than for threshold cases (37% versus 17% for binge eating remission, χ^2 [1/96] = 5.99, p = .014; 43% versus 19% for compensatory behavior remission, χ^2 [1/96] = 4.85, p = .028). At the end of the study, 63% of those with subthreshold bulimic pathology at baseline no longer met criteria for subthreshold or full threshold bulimia, 2% met criteria for full threshold bulimia, and 33% had subthreshold bulimic pathology. For those participants meeting criteria for bulimia nervosa at baseline, 38% no longer met criteria at the end of the study, 27% still met criteria, and 35% showed subthreshold bulimic pathology. [2]

[1]We considered alternative operationalizations of symptom remission (e.g., 3 or 5 months), but more participants classified as recovered with these definitions subsequently relapsed (18% and 19% respectively) than was the case for the 4-month operationalization of remission (8%). We also considered even shorter (< 3 months) or even longer (> 5 months) operationalizations of remission, but the average cell sizes for the smaller cell (i.e., persistent group or recovered group) became too small to permit adequate statistical power (M cell size of smallest cell = 17). It should be noted that previous natural history studies required symptom remission for 6-months (Stice & Agras, 1998) or 18-months (Fairburn et al., 2003), but that neither of these operattionalizations worked well in the present study.

[2]Due to low occurrence for cross-over from bulimia nervosa to anorexia nervosa, we were unable to include such cross-over in our analyses or description of course. Only one participant developed low body weight during the course of the study, but did not have additional symptoms of anorexia, such as fear of weight gain or amenorrhea.

Predicting time to remission

Beta weights, hazard ratios (HR), and confidence intervals of hazard ratios appear in Table 2. Elevated thin ideal internalization, expected reward from eating, and frequency of binge eating at baseline significantly predicted longer time to remission of binge eating behaviors. Elevated dietary restraint and frequency of compensatory behavior at baseline significantly predicted longer time to remission of compensatory behaviors. We next estimated multivariable models with predictors that showed significant prediction in the earlier models. Expected reward from food intake was the only predictor to show a significant unique effect in the model predicting binge eating remission (β =−.49, HR=1.63, p=.024). Dietary restraint was the only predictor to show a significant unique effect in the model predicting compensatory behavior remission (β=−.52, HR=1.68, p=.006).[3] Significant effects were small to moderate in magnitude.

Discussion

Results revealed high rates of recovery and relapse for bulimic pathology during this 1-year natural history study; although participants reported brief periods of symptom remission, this often did not persist. This symptom fluctuation may have been more difficult to document in past natural history studies that used 9–18 month intervals between assessments [8–9]. In addition, over half of the sample never experienced a period of remission lasting at least four months during the 1-year follow-up. A greater percentage of participants with subthreshold bulimic nervosa recovered (40%) over the 1-year follow-up compared to those with threshold bulimia nervosa (18%), suggesting that more severe levels of bulimic symptoms are more chronic. Although the 18% 1-year recovery rate we observed for threshold bulimia nervosa was similar to the 24% 1-year recovery rate observed by Milos et al. [6], our 40% recovery rate for subthreshold cases was higher than the 31% recovery rate observed for ED-NOS cases by Milos et al. It is possible that this discrepancy emerged because the ED-NOS cases were more heterogeneous or because they were from a clinical sample. Indeed, clinical samples often show longer duration of illness than non-treatment samples (11.7 years [6] and 8.3 years, [17] respectively).

Results suggested that there might be somewhat distinct maintenance factors for binge eating and compensatory behaviors. Thin ideal internalization, expected reward from food intake, and initial frequency of binge eating were all associated with a longer time to remission from binge eating. The evidence that elevated thin-ideal internalization predicts persistence of binge eating replicates the effects from the two prior natural history studies [8–9]. Thin ideal internalization may paradoxically maintain binge eating because it is an unattainable goal that promotes hopelessness and rumination about forbidden foods, which increases the risk for binge eating. The finding that initial elevations in binge frequency predicted a longer time to remission also seems to imply some self-maintaining process perpetuates binge eating. The finding that elevated expected reward from eating predicted a longer time to remission from binge eating shows conceptual convergence with the predictive effects of a history of obesity from the Fairburn et al., [8] study. This seem to imply that individuals who experience greater reward from food intake or who have a greater drive to eat, which may increase the odds of a history of obesity, are at heightened risk for persistent binge eating. These effects may be a product of abnormalities in reward neural circuitry.

[3]We conducted supplemental analyses predicting remission from both binge eating and compensatory behaviors simultaneously. Dietary restraint (B=−.19, HR=1.21, p=.009) and thin ideal internalization (B=−1.42, HR=4.13, p=.003) were the only significant predictors of time to remission of bulimic symptoms in separate analyses. Furthermore, only thin ideal internalization (B=−1.10, HR=3.01, p=.015) remained a significant predictor when both were entered into a multivariable model.

Dietary restraint predicted a longer time to remission from compensatory behaviors, replicating the results from one of the earlier natural history studies [9]. Individuals with an overeating tendency, who attempt (usually unsuccessfully; [18]) to limit their caloric intake, may begin to rely on more drastic weight control behaviors. Alternatively, it is possible that restraint scales simply identify individuals with an overeating tendency that they are trying to curb through dieting, but that it is this overeating tendency that really increases the likelihood of persistent use of compensatory behaviors. The evidence that individuals who engage in more frequent compensatory behaviors show a longer time to remission from these behaviors also seems to imply some self-perpetuating process. Perhaps once people engage in compensatory behaviors, they rely on these behaviors for some secondary gain, such as emotional catharsis, which perpetuates this behavior.

The findings from this study have implications for treatment of bulimic pathology. Interventions that reduce eating expectancies may have increased effectiveness for treatment of binge eating, as this was the only significant predictor in the multivariable model of time to remission of binge eating. Additionally, providing healthy alternatives to dieting or compensatory behaviors may help those with high dietary restraint scores refrain from using these unhealthy methods, and may be an important component for treatment. Furthermore, treatments that produce rapid reduction of binge eating and compensatory behaviors may be more successful due to the self-maintaining effects of these symptoms. Our results showed that greater frequency of binge eating and compensatory behaviors predicted greater time to remission. Thus, if these behaviors are reduced quickly, there may be greater likelihood of remission. This is congruent with prior treatment studies that have shown that rapid responders to treatment showed greater symptom control and were less likely to relapse than slower responders [19–21].

Although this is one of the only natural history studies of bulimic pathology to use a non-treatment sample and frequent diagnostic interviews, it is not without limitations. First, the small sample size limited the power to detect small effects and hindered our ability to examine diagnostic cross-over.[4] Second, use of a longer follow-up period would have allowed us to more precisely gauge that course of this disorder and average time to remission. Third, it might be informative to assess putative maintenance factors at follow-up assessments to determine whether there are reciprocal relations between the maintenance factors and bulimic symptoms. Fourth, it would have been desirable to assess potential biological maintenance factors, such as heightened reward from food intake and gastric capacity (e.g., [22]). Fifth, we did not measure treatment utilization, so we are not able to determine whether treatment impacted remission or relapse rates in the sample. Sixth, repeated assessments may affect participant's behaviors due to increased attention to their symptoms. Additionally, because bulimia nervosa tends to be secretive, it is possible that those who volunteer for a study on the course of eating pathology may differ from others with the disorder. Finally, although prospective studies are an improvement upon cross-sectional analyses, there is still the possibility that third variables account for relations between factors and remission from bulimic pathology. Randomized controlled trials could address this limitation by assigning participants to conditions that manipulate change in these maintenance factors and assess subsequent change in bulimic symptoms. Despite these limitations, the findings contribute to our understanding of potential factors that might serve to maintain this pernicious eating disorder and have the potential to inform the development of more efficacious treatments.

[4]Power analyses showed that with a sample size of 96, we had power of .85 to detect a medium effect (HR = 1.96) for all outcome measures. We calculated power for medium effects, rather than conducting separate analyses for the 18 effects in the study (the average HR for our significant effects was 2.1)

Acknowledgments

This study was supported by research grants (MH/DK61957 and MH6450), predoctoral fellowships (MH64254 and MH081588), and a career award (MH01708) from the National Institutes of Health.

References

1. Stice, E.; Bulik, CM. Eating Disorders. In: Beauchaine, TP.; Hinshaw, SP., editors. Child and Adolescent Psychopathology. Hoboken, New Jersey: Wiley and Sons; 2008. p. 643-669.
2. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. The natural course of bulimia nervosa and binge eating disorder in young women. Arch Gen Psychiat. 2000; 57:659–665. [PubMed: 10891036]
3. Fairburn CG, Norman PA, Welch SL, O'Connor ME, Doll HA, Peveler RC. A prospective study of outcome in bulimia nervosa and the long-term effects of three psychological treatments. Arch Gen Psychiat. 1995; 52:304–312. [PubMed: 7702447]
4. Grilo CM, Sanislow CA, Shea MT, Skodol AE, Stout RL, Pagano ME, et al. The natural course of bulimia nervosa and eating disorder not otherwise specified is not influenced by personality disorders. Int J Eat Disorder. 2003; 34:319–330.
5. Keel PK, Dorer D, Franko D, Jackson S, Herzog DB. Postremission predictors of relapse in women with eating disorders. Am J Psychiat. 2005; 162:2263–2268. [PubMed: 16330589]
6. Milos G, Sindler A, Schnyder U, Fairburn CG. Instability of eating disorder diagnoses: Prospective study. Brit J Psychiat. 2005; 187:573–578.
7. Newman D, Moffitt T, Caspi A, Magdol L, Silva P, Stanton W. Psychiatric disorder in a birth cohort of young adults: Prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. J Consult Clin Psych. 1996; 64:552–562.
8. Fairburn CF, Stice E, Cooper Z, Doll HA, Norman PA, O'Connor ME. Understanding persistence of bulimia nervosa: A five-year naturalistic study. J Consult Clin Psych. 2003; 71:103–109.
9. Stice E, Agras WS. Predicting onset and cessation of bulimic behaviors during adolescence: A longitudinal grouping analysis. Behav Ther. 1998; 29:257–276.
10. Crow SJ, Agras WS, Halmi K, Mitchell JE, Kraemer HC. Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: A multicenter study. Int J Eat Disorder. 2002; 32:309–318.
11. Stice E, Marti N, Spoor S, Presnell K, Shaw H. Dissonance and healthy weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. J Consult Clin Psych. 2008; 76:329–340.
12. Spangler D, Stice E. Validation of the Beliefs About Appearance Scale. Cognitive Ther Res. 2001; 25:813–827.
13. Bond MJ, McDowell AJ, Wilkinson JY. The measurement of dietary restraint, disinhibition and hunger: An examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). Int J Obesity. 2001; 25:900–906.
14. Beck AT, Steer RM, Garbin M. Psychometric properties of the Beck Depression Inventory: 25 years of evaluation. Clin Psychol Rev. 1988; 8:77–100.
15. Smith GT, Simmons JR, Flory K, Annus AM, Hill KK. Thinness and eating expectancies predict subsequent binge-eating and purging behavior among adolescent girls. J Abnorm Psychol. 2007; 116:188–197. [PubMed: 17324029]
16. American Psychological Association. Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). Washington, DC: 1994.
17. Hudson J, Hiripi E, Pope H, Kessler R. The prevalence and correlates of eating disorders in the National Comorbitity Survey Replication. Biol Psychiat. 2007; 61:348–358. [PubMed: 16815322]
18. Stice E, Cooper JA, Schoeller DA, Tappe K, Lowe MR. Are dietary restraint scales valid measures of moderate- to long-term dietary restriction? Objective biological and behavioral data suggest not. Psychol Assess. 2007; 19:449–458. [PubMed: 18085937]

19. Grilo CM, Masheb RM. Rapid response predicts binge eating and weight loss in binge eating disorder: Findings from a controlled trial of orlistat with guided self-help cognitive behavioral therapy. Behav Res Ther. 2007; 45:2537–2550. [PubMed: 17659254]

20. Olmstead MP, Kaplan AS, Rockert W, Jacobsen M. Rapid responders to intensive treatment of bulimia nervosa. Int J Eat Disord. 1996; 19:279–285. [PubMed: 8704727]

21. Mitchell JE, Pyle RL, Pomeroy C, Zollman M, Crosby R, Seim H, Eckert ED, Zimmerman R. Cogntiive-behavioral group psychotherapy of bulimia nervosa: Importance of logistical variables. Int J Eat Disord. 1993; 14:277–287. [PubMed: 8275064]

22. Geliebter A, Melton PM, McCray RS, Gallagher DR, Gage D, Hashim SA. Gastric capacity, gastric emptying, and test-meal intake in normal and bulimic women. Am J Clin Nutr. 1992; 56:656–661. [PubMed: 1414964]

Table 1

Correlations Among Predictors at T1

	2.	3.	4.	5.	6.	7.	8.	9	Mean (SD)	Range
1. Dietary restraint	−.24*	.18	−.27**	−.18	−.10	.09	−.14	.21*	14.72 (4.10)	3.6–23.5
2. Depressive symptoms		.15	.18	−.04	.12	−.01	.02	.04	1.06 (1.31)	0.1–2.76
3. Thin ideal internalization			.36***	.24*	.13	.38***	.29**	.33**	3.24 (.77)	2.9–5.0
4. Body satisfaction				.11	.38***	.02	.14	.20*	3.63 (1.13)	1.0–6.2
5. Hunger					.05	.24*	.35***	−.20	9.66 (2.85)	2.0–14.0
6. Expectation that food is rewarding						.05	.04	.17	3.06 (.26)	2.5–3.8
7. Expectation that food reduces negative mood							.28**	−.18	2.97 (.84)	1.0–5.0
8. Binge eating frequency								−.001	9.99 (9.47)	0–30
9. Compensatory symptoms frequency								--	18.95 (31.60)	0–294

Note:

* p<.05;

** p<.01;

*** p<.001

Table 2

Survival analysis predicting time to remission of binge eating and compensatory behaviors

Predictors of remission of binge eating for at least 4 months				
Predictor	**B**	**Inverse HR**	**95% CI for Hazard Ratio**	**p**
T1 Dietary restraint	−.25	1.29	.92–1.81	.14
T1 Depressive symptoms	−.52	1.70	.75–3.82	.20
T1 Thin ideal internalization	−.45	1.57	1.09–2.27	.02
T1 Body satisfaction	.04	.96	.67–1.38	.84
T1 Hunger	−.31	1.36	.98–1.88	.06
T1 Expectation that food is rewarding	−.46	1.58	1.08–2.33	.02
T1 Expectation that food reduces negative mood	−.15	1.16	.81–1.67	.41
T1 Binge eating frequency	−.61	1.84	1.05–3.23	.03
T1 Compensatory behavior frequency	−.25	1.28	.64–2.59	.49
Predictors of remission of compensatory behaviors for at least 4 months				
T1 Dietary restraint	−.56	1.75	1.21–2.49	.002
T1 Depressive symptoms	−.51	1.67	.68–4.06	.26
T1 Thin ideal internalization	−.31	1.36	.93–2.00	.11
T1 Body satisfaction	−.06	1.06	.72–1.57	.75
T1 Hunger	.07	1.07	.74–1.54	.72
T1 Expectation that food is rewarding	.17	.84	.58–1.24	.36
T1 Expectation that food reduces negative mood	−.24	1.22	.87–1.87	.22
T1 Binge eating frequency	−.27	1.31	.81–2.12	.27
T1 Compensatory behavior frequency	−1.30	3.65	1.01–13.16	.05