

A Prospective Test of the Relation Between Weight Change and Risk for Bulimia Nervosa

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ABSTRACT

Objective: Prospectively investigate whether weight gain or weight loss increases risk for onset of binge eating and purging in adolescent women.

Method: Diagnostic interviews and direct measures of body mass were completed by 496 adolescent women annually for 8 years.

Results: Substantial weight gain or weight loss during the study produced a sevenfold increase in risk for future onset of threshold or subthreshold bulimia nervosa (BN) relative to weight-stable participants, though the low incidence rate limited statistical power. Those who showed onset of threshold/subthreshold BN experienced greater increases in

weight in the 2 years before onset of their eating disorder relative to healthy comparison participants.

Discussion: This is the first prospective study to demonstrate that weight gain and weight loss may both increase risk for future onset of bulimic pathology. Results suggest that young women who have difficulty limiting their dietary intake are at increased risk for BN, an eating disorder characterized by loss of control over eating. © 2010 by Wiley Periodicals, Inc.

Keywords: bulimia nervosa; weight change; prospective studies; adolescents

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Introduction

Little is known about the relation between relative body weight and the development of BN. Russell,¹ in his classic paper that first formally identified this syndrome, noted that BN was “an ominous variant of anorexia” and that these patients, though typically presenting with “unremarkable” body weights, weighed significantly more before the development of their eating disorder. Others^{2–4} have presented evidence that individuals with BN are significantly weight-suppressed, that is, their current weight tends to be substantially below their retrospectively reported highest weight ever relative to controls. Because individuals with BN are typically in the normal weight range, this implies that many patients with BN were somewhat overweight at their previous highest weight. Similarly,

Fairburn et al.⁵ and Yates⁶ report that individuals with BN retrospectively reported a higher premorbid body mass than healthy controls and that there was a greater history of overweight in first-degree relatives of BN individuals.

On the other hand, the normal weight status of most individuals with BN also conceals a weight history that involved weighing significantly less than they currently weigh. Garner and Fairburn³ reviewed evidence suggesting that most individuals with BN lose a great deal of weight in the process of developing their disorder, with roughly a third reaching body weights characteristic of anorexia nervosa before gaining weight and transitioning to BN.⁷ Further, case study and nonexperimental research suggests that significant weight loss precedes—and presumably spurs the development of—binge eating.^{8–10} In sum, many individuals with BN report a history of both higher and lower body weights relative to their weights when they come to the attention of researchers or clinicians. However, little is known about whether weight gain or weight loss increases risk for the development of BN.

A fundamental weakness in existing studies of the possible role of weight history in BN is that the relevant data are collected retrospectively. Such retrospective reports are often inaccurate because of natural forgetting that happens over long periods of time, because memories are influenced by current mood state and subsequent events, people

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tend to distort memories in a more socially desirable direction, people may reinvent the past to suit their current needs and circumstances, and people tend to remember events as happening earlier than they actually did.^{11,12} It would be preferable to examine the relation of weight change to the development of bulimic pathology prospectively by (a) examining bulimic symptom frequency before and after individuals have gained or lost a significant amount of weight and (b) by identifying individuals who develop bulimic pathology and determining whether weight loss or weight gain preceded the emergence of this pathology relative to a similar group that did not develop bulimic pathology. No prospective study has tested whether significant weight loss or weight gain increases risk for future development of bulimic pathology or tested whether those who develop bulimic pathology experience accelerated weight loss or weight gain before the development of this pathology. Because self-reported weight and height, whether collected contemporaneously or retrospectively, have questionable accuracy,¹³ it would be preferable to rely on directly measured height and weight.

This study prospectively examined the relation between weight change and onset of threshold or subthreshold BN in two different ways. The first aim was to identify participants in this data set who experienced a substantial weight loss or weight gain during the 8 years and determine if either weight change pattern was associated with increased risk for onset of threshold/subthreshold BN in the year during or soon after the weight change occurred relative to a control group who was weight stable across the 8 years. The second aim was to test whether individuals who showed onset of threshold/subthreshold BN evidence a history of significant weight loss or weight gain previous to onset of the eating disorder relative to those who remained free of this eating disorder.

Method

Participants and Procedures

Participants were 496 adolescent girls from public and private middle schools in a large US city. These data were collected yearly for 8 years as part of a larger study.¹⁴ Adolescents ranged from 12 to 15 years of age ($M = 13$) and were in 7th or 8th grade at baseline (year 1). Pubertal development was not assessed in detail, but some individuals ($n = 127$) had not experienced menarche by the time of the baseline assessment. The sample included 2%

Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% who specified other/mixed racial heritage, which was representative of the schools from which we sampled. Average parental education, a proxy for socioeconomic status, was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was representative of the city from which we sampled.

The study was described as an investigation of adolescent mental and physical health. An active parental consent procedure was used to recruit participants, wherein an informed consent letter describing the study was sent to parents of eligible girls (a second mailing was sent to nonresponders). This resulted in an average participation rate of 56%, which was similar to rates in other school-recruited samples that used active consent procedures and structured interviews (e.g., 61%).¹⁵ Participants completed a structured interview assessing eating disorder symptoms, depression,¹⁶ and had their weight and height measured by female assessors at baseline (year 1) and at seven annual follow-ups (year 2–year 8). Female assessors with at least a bachelor's degree in psychology conducted interviews. They attended 24 h of training, wherein they received instruction in structured interview skills, reviewed diagnostic criteria for relevant DSM-IV disorders,¹⁷ observed simulated interviews, and role-played interviews. Assessors had to demonstrate an inter-rater agreement ($\kappa > 0.80$) with supervisors using tape-recorded interviews before collecting data. A randomly selected subset of interviews (5%) was recorded annually to ensure that assessors continued to show acceptable inter-rater agreement ($\kappa > 0.80$) with experts. Assessments took place at schools during or immediately after school hours or at participants' houses. Participants received a gift certificate or cash payment for completing each assessment.

In the first set of analyses, we identified adolescents who experienced either a 10% weight gain or weight loss over a 1-year period (based on age-adjusted normative weight charts showing an average increase in BMI of 2% per year) during the first 5 years of the study. Regardless of when this weight change occurred, this year was considered an index year for either weight gain or weight loss. A third group of weight stable adolescents, who never experienced a weight change greater than 5% year-to-year across the 8 years, were identified and referred to as weight stable participants. These participants' scores on a measure of bulimic symptoms were then compared at the end of the year when the weight change occurred and for the two subsequent yearly assessments. This allowed us to determine if a large increase or decrease in body mass increased the risk of subsequently developing threshold/subthreshold BN relative to a weight stable

TABLE 1. Group membership and attrition

Study Year	Assessed		Not Assessed		Weight Losers Identified		Weight Gainers Identified		BN Spectrum Identified	
1	496	(100%)	0	(0%)						
2	482	(97.2%)	13	(2.6%)	13	(2.7%)	30	(6.2%)		
3	477	(96.2%)	18	(3.6%)	16	(3.4%)	23	(4.8%)	0	(0%)
4	486	(98.0%)	9	(1.8%)	19	(3.9%)	10	(2.1%)	5	(1.0%)
5	480	(96.8%)	15	(3.0%)	14	(2.9%)	9	(1.9%)	5	(1.0%)
6	431	(86.9%)	34	(6.9%)	14	(3.3%)	14	(3.3%)	7	(1.6%)
7	452	(91.1%)	43	(8.7%)					4	(0.9%)
8	451	(90.9%)	44	(8.9%)						

Weight losers, participants with a 10% age-adjusted BMI decrease; Weight gainers, participants with a 10% age-adjusted BMI increase; BN spectrum, participants who met criteria for clinical or subclinical bulimia nervosa.

control group. In the second set of analyses, individuals who showed onset of threshold or subthreshold BN after year 2 were identified. Then, the weight change trajectory experienced by this group during the two annual visits preceding the onset of bulimic pathology was calculated. These weight changes were compared with those of all other participants who never showed onset of bulimic pathology. In this way, we were able to determine if the bulimic pathology group experienced a different 2-year weight trajectory before their identification than did the nondisordered control group.

Measures

Eating Pathology. The Eating Disorder Diagnostic Interview,¹⁸ a semistructured interview that was adapted from the Eating Disorder Examination,¹⁹ assessed DSM-IV criteria for anorexia nervosa (AN), BN, and binge eating disorder (BED) at each of the eight annual assessments. These data also allowed us to determine whether participants met criteria for threshold or subthreshold BN at each assessment point. In accordance with previous studies,^{18,20,21} participants who endorsed symptoms from each domain for BN, but who endorsed a subthreshold level on at least one symptom were given subthreshold diagnoses. For a threshold BN diagnosis, we required participants to report at least 24 uncontrollable binge eating episodes and 24 compensatory behavior episodes (self-induced vomiting, laxatives or diuretics, fasting, and excessive exercise intended to compensate for overeating) over a 3-month period, and to report that weight and shape was definitely one of the main aspects of self-evaluation. For a subthreshold BN diagnosis, we required participants to report at least 6 uncontrollable binge eating episodes and 6 compensatory behavior episodes over a 3-month period, and to report that weight and shape was definitely an aspect of self-evaluation. Excessive exercise was defined as at least 1 h of vigorous exercise or 2 h of moderate exercise that was engaged in specifically to compensate for a binge eating episode. Fasting was defined as abstaining from caloric intake (meals or snacks) for ~24 h or more for the purpose of

weight control (e.g., only eating dinner for 2 consecutive days).¹⁷

To assess the test-retest reliability for eating disorder diagnoses for this adapted interview, a randomly selected subset of 137 participants who were interviewed by the assessors for this study and another ongoing study¹⁸ were reinterviewed by the same assessor within a 1-week period, resulting in high test-retest reliability for threshold and subthreshold diagnoses of AN, BN, or BED ($\kappa = 0.96$). To assess the inter-rater agreement for these threshold and subthreshold eating disorder diagnoses, a randomly selected subset of 149 participants who were interviewed by the assessors for this study and the other ongoing study were reinterviewed by a second blinded assessor, resulting in high inter-rater agreement ($\kappa = 0.86$). The EDDI has also been shown to be sensitive to detecting intervention effects and has shown predictive validity for future onset of depression in past studies.^{16,18,22}

Body Mass. The body mass index (BMI = kg/m^2) was used to reflect relative weight.²³ Height was measured to the nearest millimeter using portable stadiometers. Weight was assessed to the nearest 0.1 kg using a SECA scale, model 770 with the girls wearing light indoor clothing without shoes or coats. Two measures of height and weight were obtained and averaged. The BMI shows convergent validity ($r = .80-.90$) with direct measures of body fat such as dual energy X-ray absorptiometry.²³

Results

Classification of Participants

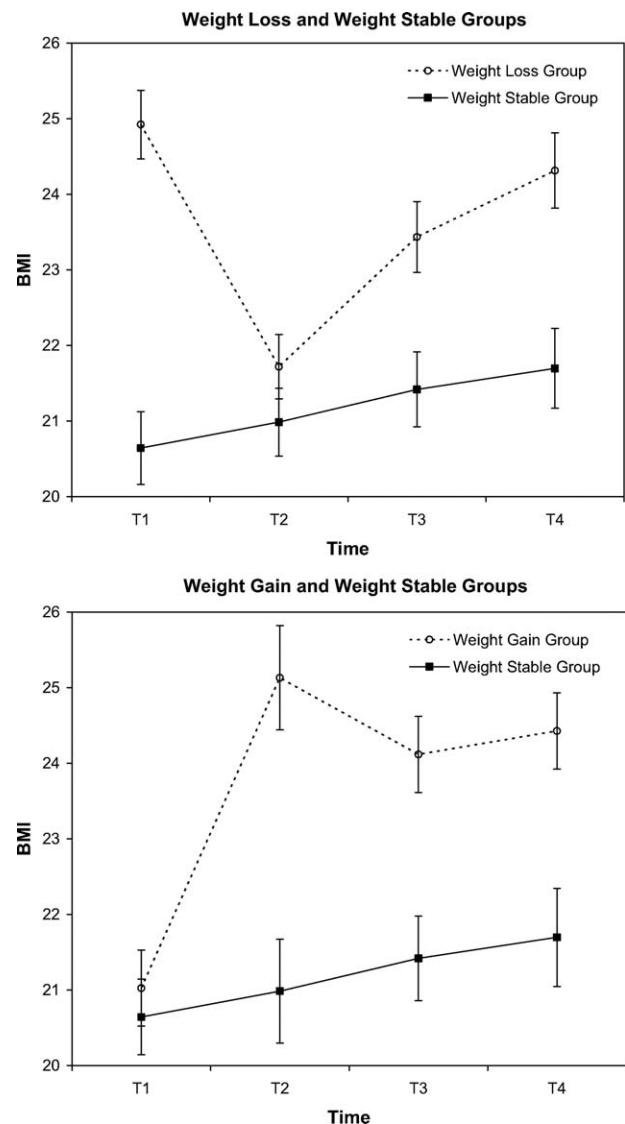
The number of participants providing data at each assessment is reported in **Table 1**. An attempt was made to assess each participant yearly, even if she missed a previous assessment. Participants missing data at any assessment point did not differ significantly from the remaining participants on

demographic factors or any of the study variables, suggesting that attrition should not introduce bias.

As the goal of the first analysis was to investigate risk for onset of threshold/subthreshold BN during or after a considerable weight gain or loss, participants were identified who experienced a 10% age-adjusted weight increase or decrease during any of the first five 1-year intervals of the study. The number of participants who showed a 10% change in age-adjusted BMI at each of these intervals is reported in **Table 1**. Because an average of only 17 participants per year showed a 10% age-adjusted decrease, and an average of 18 participants showed a 10% age-adjusted increase, in BMI scores over each of the 1-year intervals, we pooled participants who showed a 10% age-adjusted weight change across any of the five intervals (year 1 to year 2, year 2 to year 3, year 3 to year 4, year 4 to year 5, or year 5 to year 6) to maximize statistical power. In total, there were 84 adolescent girls who evidenced a 10% age-adjusted reduction in BMI over any of the five 1-year detection intervals in this study, though only 76 of these individuals provided interview data at 1 and 2 years following their weight loss. Similarly, there were 92 adolescent girls who evidenced a 10% age-adjusted weight increase in BMI over any of the five 1-year detection intervals, but only 86 of them had 2 years of follow-up data. For the pooled data, the two assessments that encompass the 10% age-adjusted BMI change are referred to as T1 and T2. The two annual assessments that follow the BMI change are referred to as T3 and T4. A similar procedure has been used in previous research.²⁴

A third group of “weight stable” participants was created, which consisted of individuals who experienced no more than a 5% age-adjusted change in BMI across any of the seven 1-year intervals of the study. Eighty-five participants were identified as “weight stable” across the 8-year study. This number is likely an underestimate of the true number of weight stable individuals, as only participants who had complete data from all eight yearly assessments were considered eligible for the weight stable group. This was done to avoid including participants who may have experienced a 10% weight change during an interval during which they were not assessed. As the participants in the weight loss and weight gain groups could have been identified at any of the first five 1-year intervals of the study, the detection interval for the weight stable participants was randomly assigned from one of the first 5 years of the study to maintain equivalence among detection intervals for the three groups. Analyses confirmed that the weight stable group did not dif-

FIGURE 1. BMI trajectory of the weight loss group (participants with a 10% age-adjusted BMI decrease) and the weight gain group (participants with a 10% age-adjusted BMI increase) compared with the weight stable group (no year-to-year weight change greater than 5% across the 8 year study). Participants in the two weight change groups were pooled by fixing the year of weight change at T1 to T2. Points represent mean BMI; vertical lines depict standard errors of the means.



fer significantly from the weight gain and weight loss groups on demographic factors.

Change in BMI by Group

Weight Loss Group. The overall trend for the weight loss group compared with the weight stable group is depicted in the top panel of **Figure 1**. As expected given the strategy used to create the comparison groups, the weight loss group showed significantly greater decreases in BMI over the 1-year detection interval relative to the weight stable group, as indi-

cated by a significant Time by Group interaction in a repeated measures analysis of variance model ($F(1, 159) = 330.00, p < .001, \eta_p^2 = 0.68$). Post hoc within subjects t -tests revealed a significant decrease in BMI in the weight loss group ($M = -3.20, SD = 1.64; t(75) = 17.00, p < .001, \eta_p^2 = 0.79$), and a comparatively small, but statistically significant BMI increase in the weight stable group ($M = 0.34, SD = 0.70; t(84) = 4.54, p < .001, \eta_p^2 = 0.20$). (A change of one BMI unit equals ~ 4 – 9 lbs, depending on height). At the initial assessment of the detection interval, the BMI of the weight loss group ($M = 24.92, SD = 4.50$) was significantly higher than the BMI of the weight stable group ($M = 20.64, SD = 3.88; t(159) = 6.48, p < .001, \eta_p^2 = 0.21$). However, at the assessment following the weight change, there was no significant difference in BMI between the weight loss group ($M = 21.72, SD = 3.72$) and the weight stable group ($M = 20.99, SD = 4.09; t(159) = 1.19, p = .238, \eta_p^2 = 0.01$). Over the two, 1-year intervals following the weight change, the weight loss group showed significantly greater increases in BMI ($M = 2.60, SD = 2.99$) than were shown in the weight stable group ($M = 0.71, SD = 1.08$), as indicated by a significant Time by Group interaction in a repeated measures analysis of variance model ($F(2, 318) = 14.75, p < .001, \eta_p^2 = 0.09$). Post hoc t -tests indicated no difference between the two groups on BMI at the assessment directly following the weight change (as described above). However, the weight loss group exhibited a significantly higher BMI than the weight stable group at both yearly assessments following the BMI decrease ($p < .005$ for both comparisons). In sum, before experiencing weight loss, the weight loss group was significantly heavier than the weight stable group. After losing weight, the BMI of the weight loss group was equivalent to the BMI of the weight stable group. However, members of the weight loss group tended to regain the majority of the weight that was lost over the following 2 years.

Weight Gain Group. The overall trend for the weight gain group compared with the weight stable group is depicted in the bottom panel of **Figure 1**. Again, as expected given the strategy used to create the comparison groups, the weight gain group showed significantly greater increases in BMI over the 1-year reference period than were shown in the weight stable group, as indicated by a significant Time by Group interaction in a repeated measures analysis of variance model ($F(1, 169) = 41.25, p < .001, \eta_p^2 = 0.20$). A post hoc within subjects t -test revealed a significant increase in BMI in the weight gain group ($M = 4.11, SD = 5.36; t(85) = 7.11, p < .001, \eta_p^2 = 0.37$). As noted, there was a small but

statistically significant BMI increase in the weight stable group over the same period. At the assessment preceding the weight change interval, the BMI of the weight gain group ($M = 21.03, SD = 5.28$) was not significantly different than the BMI of the weight stable group ($M = 20.64, SD = 3.88; t(169) = 0.54, p = .591, \eta_p^2 = 0.002$). However, at the assessment following the weight change, there was a significant difference between the BMI of the weight gain group ($M = 25.13, SD = 8.00$) and the weight stable group ($M = 20.99, SD = 4.09; t(169) = 4.26, p < .001, \eta_p^2 = 0.10$). Over the two 1-year intervals following the weight change, there was a significant difference in the trajectory of weight change between the two groups, as evidenced by a significant Time by Group interaction in a repeated measures analysis of variance model ($F(2, 338) = 4.15, p = .017, \eta_p^2 = 0.02$). A Post hoc t -test showed a small, but statistically significant, increase in BMI in the weight stable group in the 2 years following the detection interval ($M = 0.71, SD = 1.08; t(84) = 6.06, p < .001, \eta_p^2 = 0.26$). The weight gain group showed a nonsignificant weight decrease over the same period ($M = 0.70, SD = 6.12; t(85) = 1.07, p = .195, \eta_p^2 = 0.02$). The weight gain group exhibited a significantly higher BMI than the weight stable group at both yearly assessments following the BMI increase ($p < .005$ for both comparisons). In sum, before experiencing weight gain, the BMI of the weight gain group is equivalent to the BMI of the weight stable group. After gaining weight, the BMI of the weight gain group is significantly greater than that of the weight stable group and tends to remain at that high level over the following 2 years.

Onset of Threshold/Subthreshold BN by Group. We next tested whether the two weight-change groups showed differential onset of threshold/subthreshold BN relative to the weight stable group. As in the case of the BMI change analyses, the detection interval and the 2 following years were examined separately. First, the three groups were probed for participants who developed threshold or subthreshold BN at any time during the study. In the weight loss group, eight such participants were identified (10.5% of weight losers); one was threshold, seven were subthreshold (one progressed to threshold at a later assessment). Two of these cases first met criteria before the detection interval (the average time of BN onset was 1.5 years before the 10% weight loss), and six first met criteria following the detection interval (the average delay in BN onset was 3 years after the 10% weight loss). A further eight participants meeting criteria for onset of threshold/subthreshold BN were identified in the weight gain group (9.3% of weight gainers); all were subthres-

hold (two progressed to threshold at a later assessment). One of these cases first met criteria 3 years before the detection interval, two cases were identified during the detection interval, and five cases were identified following the detection interval (the average delay in BN onset for these five cases was 4.6 years after the 10% weight gain). One case of subthreshold BN was identified in the weight stable group (1.2% of weight stables). The relation with the detection interval is not reported for this case, as the detection interval was randomly assigned for members of the weight stable group.

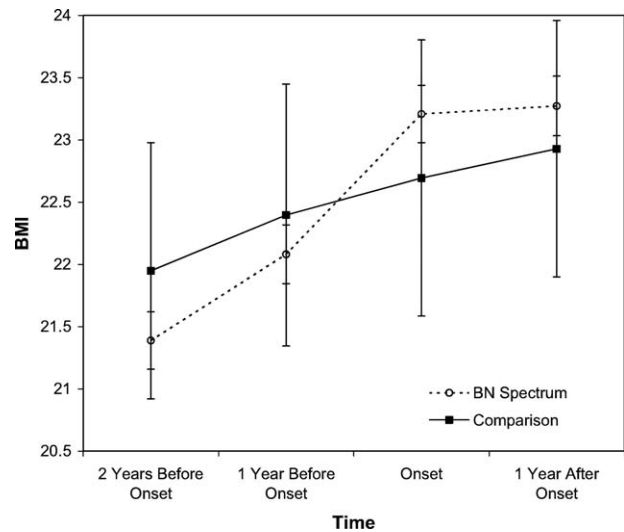
Fisher's exact test was used to determine whether an association exists between weight change group and frequency of diagnosis with threshold or subthreshold BN. Cases from the two weight change groups were included in these analyses only if they were first diagnosed during or after the 10% age-adjusted weight change. Cases in which the disorder was diagnosed before the weight change were eliminated from these analyses. Threshold or subthreshold BN was identified in 8.1% of the weight loss group, compared with 1.2% of the weight stable group (Odds Ratio = 7.41, $p = .39$, Fisher's exact test). Likewise, the disorder was identified in 8.2% of the weight gain group, compared with 1.2% of the weight stable group (Odds Ratio = 7.54, $p = .32$, Fisher's exact test).

Weight Change Preceding Onset of BN

Twenty-seven participants were identified who first showed onset of threshold/subthreshold BN during years three through seven of the study. Of those, 21 had weight information available for 2 years preceding, and one year following, the onset of bulimic pathology (see Table 1 for the number of participants detected in years 3 through 7). All 21 cases were subthreshold (four progressed to threshold at a later assessment). Participants who met criteria for bulimic pathology during years 1, 2, and 8 would have had insufficient data for the following analyses and were excluded. As in the previous analyses, all participants who were diagnosed with bulimic pathology during years 3 to 7 were pooled to increase statistical power. The rates of other threshold and subthreshold eating disorders in this sample have been reported elsewhere.²⁵

A non-BN group was created that included all of the participants with adequate weight data that did not show onset of threshold/subthreshold BN ($n = 420$) at any time during the study. For the bulimic pathology group, participants had to show onset during years 3 through 7. Therefore, the detection

FIGURE 2. BMI trajectory of the BN spectrum group (participants who met criteria for subclinical Bulimia Nervosa) relative to year of first diagnosis, compared with the comparison group (all other participants not meeting a diagnosis of a BN spectrum disorder during the study). Points represent mean BMI; vertical lines depict standard errors of the means.



year for the participants in the non-BN group was randomly assigned from years 3 through 7.

The overall trend for the bulimic pathology group compared with the non-BN group is depicted in Figure 2. The former showed significantly greater increases in BMI over the 2 years preceding the onset of bulimic pathology relative to the comparison group, as indicated by a significant Time by Group interaction in a repeated measures analysis of variance model ($F(2, 878) = 3.72, p = .008, \eta_p^2 = 0.01$). There was no difference in weight trajectory for the two groups in the year following onset of bulimic pathology ($F(1, 439) = 0.16, p = .686, \eta_p^2 < 0.01$). The BMI of the bulimic pathology group was not significantly different from the comparison group at any of the yearly assessments ($p > .55$ for all comparisons). Thus, in the 2 years before experiencing the onset of bulimic pathology the BMI of the bulimic pathology group increased at a greater rate than that of the noneating disordered comparison group. After the onset of bulimic symptoms, the BMI of both the bulimic pathology and comparison group increased at an equivalent rate.

Discussion

Research on the relation between weight change and BN has been limited by its reliance on retro-

spective data, which have questionable validity. This is the first study to use prospective data to evaluate if a large weight loss or weight gain increases risk for onset of threshold/subthreshold BN. Overall, there was a sevenfold increase in risk for onset of bulimic pathology for participants who had gained or lost at least 10% of their weight compared with participants who were weight stable. The low incidence of BN onset resulted in low power to detect statistically significant effects in this analysis. Nevertheless, the effect sizes are quite large.

This is also the first prospective study to suggest that weight gain typically occurs before BN onset. Specifically, participants who showed an onset of threshold or subthreshold BN, compared with those who never met criteria for a diagnosis, showed significantly greater increases in BMI over the 2 years preceding the onset of the diagnosis. In the 2 years following the diagnosis, the BMI change in the bulimic pathology group did not differ significantly from that of the comparison group.

These data indicate that many participants who developed bulimic pathology experienced a significant increase in weight in the 2 years before the onset of their BN symptoms. This finding raises that possibility that young women who have difficulty limiting their dietary intake are also at increased risk for BN, an eating disorder that is characterized by overeating and a loss of control of eating. The effect sizes (though not the statistical significance) of other analyses also indicate that for some participants, substantial weight loss is associated with concurrent or subsequent onset of BN. Young women who experience a large weight loss often have a history of being overweight, so it is possible these participants shared the same vulnerability to overeating, which was temporarily controlled, presumably by strict dieting. In fact, results indicated that in the 2 years before the 10% age-adjusted BMI change, the BMIs of the weight loss group and the weight stable group accelerated significantly faster than that of the weight gain group. There was no significant difference in the BMI trajectory of the weight loss group and the weight stable group in the two years before the 10% age-adjusted BMI change. In sum, we can conclude that the onset of BN symptoms is associated with elevated BMI and substantial weight fluctuation. It is possible that weight stability itself offers protection against the risk of BN onset, or that particular characteristics of young women who remain weight stable throughout adolescence offer some protection from risk (e.g., caloric intake is motivated by homeostatic need rather than by hedonic desire).

There are several possible interpretations of the present findings. One possibility is that marked weight loss or weight gain disconnects homeostatic feeding controls and increases risk for hedonic eating that may drive binge episodes. A second possibility is that the two weight change groups were in the process of developing different weight-related problems. Most studies that have compared the onset of dieting and binge eating in individuals with BN have found that dieting (and, presumably, weight loss) precedes the development of binge eating by 1–2 years,^{26–29} whereas studies of obese individuals who engage in binge eating find that the onset of dieting and binge eating occur at about the same time, with dieting following binge eating about as often as it precedes it.^{30–32} In this study, elevated bulimic symptoms in the group that lost weight may signal the development of threshold or subthreshold BN, whereas the elevated symptoms in the group that gained weight (and the weight gain that preceded the development of BN in those that developed it) may reflect the early phases of developing overweight and threshold or subthreshold BED. In the latter case, any compensatory behaviors this group used initially to counter the effects of binge eating would presumably fade over time. A third possibility is that all three findings from this study reflect weight gain proneness among those susceptible to the development of a bulimic-spectrum disorder. That is, those who experienced BN symptoms after a weight loss (first graph in Fig. 1) had significantly higher BMIs than controls before losing weight, those who experienced BN symptoms after a weight gain (second graph in Fig. 1) had significantly higher BMIs than controls following their weight gain, and those selected based on the first emergence of BN symptoms (Fig. 2) experienced a significantly larger weight gain in the year before developing BN symptoms than the control group. From this perspective, a “primary defect” that makes certain young women susceptible to the development of BN may be their propensity toward weight gain.³³ Many of those who gain weight may work assiduously to lose it; such weight loss may coincide with, but be causally unrelated to, the development of BN, or it could combine synergistically with a propensity toward weight gain to trigger the binge-purge cycle. Additional research, preferably using prospective designs, will be needed to evaluate these alternative explanations for the current findings.

This study makes an important contribution to the literature for two reasons. First, information on weight change and onset of bulimic pathology was collected prospectively. The participants ranged in age from 12 to 15 years at year 1 and 19 to 22 years

at year 8, so they were evaluated during the period of highest risk of the onset of BN.²⁵ The sample was sufficiently large to capture the onset of disordered eating in many participants. Bulimic symptoms were assessed with a clinical interview, the EDDI, which appears to be a reliable and valid measure. Second, this is the first prospective study to show that, on average, young women gain weight before BN onset. This finding provides contrast to previous (primarily retrospective) research, and the typical conceptualization of the development of BN, which emphasize the importance of weight loss before BN onset.

Limitations of the study include a moderate participant recruitment rate and the possibility that the number of individuals who showed onset of bulimic pathology was too small to detect statistical significance in some analyses. Also, it is not possible to document that the participants who lost weight did so because they were dieting, because self-report measures of dietary restriction do not identify individuals who restrict their dietary intake.³⁴ However, previous research has demonstrated that young women who experience significant weight loss typically do so because of dieting, not for other reasons, such as health problems.³⁵

In sum, this study indicates that both substantial weight loss and weight gain are likely to precede the development of threshold and subthreshold bulimia nervosa in female adolescents. On one hand, these findings indicate that large weight fluctuations are an important feature of—and a possible contributor to—bulimia nervosa. On the other hand, it is quite challenging to try to understand how opposite weight change patterns could produce the same pathological outcome. Additional prospective research studies are needed to replicate these findings. Experimental research that promotes weight stability, especially weight gain prevention, in adolescent girls also would provide more information about the causal link between weight stability of BN risk.

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