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Fasting Increases Risk for Onset of Binge Eating and Bulimic Pathology: A 5-Year Prospective Study

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Abstract

Although adolescent girls with elevated dietary restraint scores are at increased risk for future binge eating and bulimic pathology, they do not eat less than those with lower restraint scores. The fact that only a small proportion of individuals with elevated dietary restraint scores develop bulimic pathology suggests that some extreme but rare form of dietary restriction may increase risk for this disturbance. We tested the hypothesis that fasting (going without eating for 24-hours for weight control) would be a more potent predictor of binge eating and bulimic pathology onset than dietary restraint scores using data from 496 adolescent girls followed over 5-years. Results confirmed that only 23% of participants with elevated dietary restraint scores reporting fasting. Furthermore, fasting generally showed stronger and more consistent predictive relations to future onset of recurrent binge eating and threshold/subthreshold bulimia nervosa over 1- to 5-year follow-up relative to dietary restraint, though the former effects were only significantly stronger than the latter for some comparisons. Results provide preliminary support for the hypothesis that fasting is a stronger risk factor for bulimic pathology than is self-reported dieting.

Keywords

dietary restraint; binge eating; bulimia nervosa

Theorists posit that dieting increases risk for binge eating and bulimia nervosa onset. Dieting refers to intentional restriction of caloric intake for the purposes of weight loss (Wilson, 2002). According to Polivy and Herman (1985), “Successful dieting produces weight loss, which in turn might create a state of chronic hunger, especially if such weight loss leaves the dieter at a weight below the set-point weight that is defended physiologically” (p. 196). They also postulate that a reliance on cognitive controls over eating leaves dieters vulnerable to uncontrolled eating when these cognitive processes are disrupted. In support, relative to their non-dieting counterparts, adolescent girls with elevated dietary restraint scores are at increased risk for future bulimic symptom onset (Neumark-Sztainer et al., 2006; Stice, Killen, Hayward, & Taylor, 1998), increases in bulimic symptoms (Johnson & Wardle, 2005; Stice, 2001; Wertheim et al., 2001), and threshold and subthreshold bulimia nervosa onset (Killen et al., 1996) over 1–4 year follow-up periods. However, randomized trials indicate that assignment to a weight loss diet, versus an assessment-only control condition, results in decreases in binge

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eating for overweight women (Goodrick, Poston, Kimball, Reeves & Foreyt, 1998; Klem, Wing, Simkin-Silverman, & Kuller, 1997) and decreases in bulimic symptoms for normal weight young women (Groesz & Stice, 2007; Presnell & Stice, 2003) and women with threshold and subthreshold bulimia nervosa (Burton & Stice, 2006).

These contradictory findings are troubling because they have opposing public health implications. If dieting increases bulimic symptoms, prevention and treatment interventions should seek to decrease dieting and alternative non-dieting treatments for obesity should be developed. However, if dieting reduces bulimic symptoms, prevention and treatment interventions should help individuals diet more effectively, which may reduce both bulimic pathology and obesity. Thus, we feel it is critical to investigate why these inconsistent findings emerged.

One explanation for the insistent findings, wherein individuals with elevated dietary restraint scores are at increase risk for future bulimic pathology, but assignment to an energy deficit diet reduces bulimic symptoms, is that the dietary restraint measures used in the prospective studies do not identify people on energy deficit diets. Indeed, studies using objective measures of intake (e.g., doubly labeled water) show that individuals with elevated scores on dietary restraint scales, relative to those with lower scores, do not consume significantly fewer calories during single eating episodes, multiple eating episodes, or 2–12 week periods (Martin et al., 2005; Stice, Cooper, Schoeller, Tappe, & Lowe, 2007; Stice, Fisher, & Lowe 2004; Sysko, Walsh, Schebendach, & Wilson, 2005).

The evidence that placing individuals on negative energy diets reduces bulimic symptoms and that individuals with elevated scores on dietary restraint scales are not on an energy deficit diet implies that energy deficit dieting does not cause bulimic pathology. However, given that threshold and subthrehold bulimia nervosa emerges in fewer than 5% of young women, but 40–60% of adolescent girls report weight loss dieting (Lewinsohn, Streigel-Moore, & Seeley, 2000; Neumark-Sztainer et al., 2006), it is possible that some particularly extreme weight control behavior used by a subset of individuals who report dieting could cause bulimic symptom onset.

Experiments indicate that rodents assigned to extreme caloric deprivation conditions (in which they loose 5–20% of their weight) consumed significantly more calories *ad lib* immediately after deprivation than non-deprived rodents, but that shorter caloric restriction periods do not result in elevated caloric intake (Borer, Rowland, Mirow, Borer, & Kelch, 1980; Hagan, Chandler, Wauford, Rybak, & Oswald, 2003). These findings imply that fasting (complete abstinence from caloric intake for 24 hours or more for weight control purposes), rather than more moderate dietary restriction, may increase risk for binge eating and bulimic pathology. It is vital to determine which forms of dietary restriction increase risk for bulimic pathology, so that public health professionals could provide more accurate advice regarding which behaviors may increase risk for bulimic pathology. Consequently, we tested the hypothesis that fasting is a more potent predictor of future binge eating and bulimic pathology onset than elevated scores on a widely used dietary restraint scale.

Methods

Participants and Procedures

Participants were 496 adolescent girls from middle schools who ranged in age from 11 to 15 (M = 15.4, SD = 0.7). The sample was composted of 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% other/mixed, which was representative of the sampling frame (2% Asian/Pacific Islanders, 8% African Americans, 65% Caucasians, 21% Hispanics, 4% other or mixed). Average parental education

was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was also representative of the sampling frame (34% high school graduate or less, 25% some college, 26% college graduate, 15% graduate degree). The 1-year prevalence rates of major depression (4%), bulimia nervosa (2%) and substance abuse (7%) were similar to the prevalence rates from other epidemiological studies (Lewinsohn et al., 1993). The study was described as an investigation of adolescent mental and physical health. An informed consent letter and a stamped self-addressed return envelope were sent to parents of eligible girls, resulting in an average participation rate of 56%. This is comparable to rates in other school-recruited samples that used active consent procedures and structured interviews (Lewinsohn et al., 2000). Participants completed a survey and an interview at baseline (T1) and at five annual follow-ups (T2, T3, T4, T5, & T6). Female assessors with at least a bachelor's degree in psychology attended 24 hours of training, wherein they were taught structured interview skills, reviewed diagnostic criteria for relevant disorders, observed simulated interviews, and role-played interviews. Assessors had to demonstrate an inter-rater agreement ($\kappa > .80$) with their supervisor using tape-recorded interviews before collecting data. Interviews were recorded periodically during the study to ensure that assessors showed acceptable inter-rater agreement ($\kappa > .80$).

Measures

Binge eating and bulimic pathology—The Eating Disorder Diagnostic Interview (EDDI; Stice, Marti, Spoor, Presnell, & Shaw, 2008), a semi-structured interview, assessed eating disorder symptoms on a month-to-month basis over the past year at each assessment. These data allowed us to examine predictors of future onset of recurrent binge eating (at least 2 episodes) and onset of future threshold DSM-IV bulimia nervosa or subthreshold bulimia nervosa (which would warrant a diagnosis of Eating Disorder Not Otherwise Specified per DSM-IV) during the 5-year follow-up. Participants could show onset of either outcome at any time over the 5-year follow-up because we collected monthly data on symptoms. Fasting was not considered a compensatory behavior for diagnoses. Following Stice et al. (2008), for subthreshold bulimia nervosa we required participants to report at least 6 uncontrollable binge eating episodes and 6 compensatory behavior episodes over any 3-month period (an average of twice monthly for each, versus twice weekly for a threshold diagnosis), and to report that weight and shape was definitely an aspect of self-evaluation. Those who met this definition of subthreshold bulimia nervosa in an independent sample reported significantly more functional impairment and treatment seeking relative to those free of an eating disorder and relative to themselves before showing onset of subthreshold bulimia nervosa (Stice et al., 2008), suggesting that this definition captures clinically meaningful pathology. The EDDI has shown 1-week test-retest reliability ($\kappa = .96$) and inter-rater agreement ($\kappa = .86$) for eating disorder diagnoses, as well as predictive validity and sensitivity to detecting intervention effects (Burton & Stice, 2006; Stice, Burton et al., 2004; Stice et al., 2008).

Fasting—The diagnostic item assessing frequency of fasting from the EDDI was used to determine whether participants endorsed fasting over the past year. Participants were required to be completely abstinent from caloric intake (meals or snacks) for approximately 24 hours or more to compensate for weight gain resulting from overeating; American Psychiatric Association, 1994).[1] Although most of the fasting episodes that were coded were longer than 24 hours in duration, if a participant went nearly 24 hours between eating episodes (e.g., consumed dinner at 7:00 PM one day and did not eat anything until they consumed dinner at 7:00 PM the subsequent day), this was coded as a fasting episode if it was done for weight

[1]Although the EDDI question assessing the frequency of fasting behavior describes fasting as skipping at least two meals in a row for the purposes of weight control, interviewers were trained to only code a fasting episode if the participant did not eat *any meals or snacks* for a period of approximately 24 hours or longer.

control purposes. Because this variable was highly skewed, we applied a $\log_{10}$ normalizing transformation. Given that even after transformation this variable was skewed (skew coefficient = 2.5) we also created a dichotomous variable reflecting whether participants engaged in one or more fasting episodes over the past year. Data from a randomly selected subset of 149 participants from this and another study indicated that the fasting questions showed inter-rater agreement (ICC = .79). The 1-month test-retest reliability for the total number of fasting episodes over the past month was r = .54 among 481 adolescent girls (see Stice et al., 2008 for study details). The 1-year test-retest reliability from T1 to T2 was .43 and .40 (Spearman correlations) for the continuous and dichotomous fasting variable respectively.

Dietary Restraint—The Dutch Restrained Eating Scale (van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) was designed to assess actual dietary restraint because extant dietary restraint measures did not correlate inversely with caloric intake. Participants indicate the frequency of dieting behaviors using 5-point scales ranging from *never* to *always* (items were averaged). This scale has shown internal consistency (α = .95), 2-week test-retest reliability (r = .82), and was shown to correlate negatively with self-reported caloric intake to establish that it is a valid measure of dietary restriction (Laessle, Tuschl, Kotthaus, & Pirke, 1989; van Strien et al., 1986). However, this scale does not correlate with objectively measured caloric intake (Stice, Fisher et al., 2004). We also performed a median split on this variable because this is commonly done in the literature and allowed us to investigate the predictive effect of this operationalization of dietary restraint. The 1-year test-retest reliability from T1 to T2 was .62 and .48 for the continuous and dichotomous dietary restraint variables respectively.

Results

Of the initial 496 participants, 94–99% provided data at the various follow-up assessments. The 9% of participants with missing data at any assessment did not differ significantly from the remaining participants on any of the study variables, suggesting attrition did not introduce bias.

Only 11% of the sample endorsed fasting over the past year at T1, but they reported an average of 33 fasting episodes during the year (SD = 55). They also had a significantly (F [1/475] = 4.2, p = .041, η^2 = .009) higher body mass index (BMI = kg/m^2) than those who did not report fasting (M BMI = 23.3 versus 21.9). However, mixed models indicated that the continuous and dichotomous fasting variables at T1 did not predict change in BMI over the full 5-year follow-up period (p-values .61 and .96 respectively). indicating that fasting was not associated with weight loss. The continuous dietary restraint scale was moderately correlated with the continuous fasting variable (r = .42) and the median-split dietary restraint variable was moderately correlated with the dichotomous fasting variable (r = .33). As expected, the vast majority of those who report fasting were above the dietary restraint median (91%); versus 9% of those below this median. From the alternative perspective, 23% of individuals above the dietary restraint median reported fasting, relative to 2% below this median.

Thirty-eight participants (8%) showed onset of recurrent binge eating over the 5-year follow-up: 5 showed onset between T1 and T2, 7 showed onset between T2 and T3, 12 showed onset between T3 and T4, 7 showed onset between T4 and T5, and 7 showed onset between T5 and T6 (7 participants reported recurrent binge eating at T1). Twenty-three participants (5%) showed onset of threshold/subthreshold bulimia nervosa over the follow-up period: 4 showed onset between T1 and T2, 5 showed onset between T2 and T3, 8 showed onset between T3 and T4, 4 showed onset between T4 and T5, and 2 showed onset between T5 and T6 (5 participants met criteria for subthreshold bulimic pathology at T1).

We first estimated logistic regression models to test whether fasting and dietary restraint at T1 predicted onset of recurrent binge eating and onset of threshold/subthreshold bulimia nervosa over the 5-year follow-up because this corresponds to the models used in the prospective etiologic studies which indicated that elevated dietary restraint predicted onset of bulimic pathology (e.g., Field et al., 1999; Killen et al., 1994, 1996; Pattoon et al., 1999; Stice et al., 1998). The primary purpose of this study was to investigate why these prospective studies produced findings that are inconsistent with results from experiments that manipulated dietary restraint, thus we felt it important to conduct prospective models that correspond to the analyses used in those studies. We excluded participants who reported recurrent binge eating or threshold/subtheshold bulimia nervosa at T1 (respectively) in these models to ensure that the effects were truly prospective. The continuous fasting and dietary restraint variables were standardized into z-score format to facilitate interpretation of odds ratios. Whereas the continuous and dichotomous fasting variable at T1 predicted onset of recurrent binge eating and bulimia nervosa, the continuous and dichotomous dietary restraint scales did not (Table 1). For example, although 4% of individuals who denied fasting at T1 showed onset of bulimic pathology, 13% of those who endorsed fasting at T1 showed bulimic pathology onset.

We also estimated proportional hazard models with time-varying fasting and dietary restraint measures that were lagged by one year as predictors because the null findings for dietary restraint in the above models might have emerged because the causal effect occurs over a shorter time interval (past prospective studies that produced significant effects used 1–4 year follow-up periods). These models predict onset of a putative outcome as a function of a predictor that changes across time and thus evaluate the extent to which fasting and dietary restraint measures predict the onset one year later. It should be noted that these models remove participants who show onset of recurrent binge eating or bulimic pathology after they show onset (i.e., are right censored) to ensure truly predictive effects. Both the continuous and the dichotomous fasting variables and the continuious dietary restraint variable predicted onset of recurrent binge eating and bulimic pathology over the subsequent 1-year period (Table 1). The dichotomous dietary restraint variable was not significantly related to either outcome.

Discussion

This report tested the hypothesis that fasting was a more potent predictor of future onset of recurrent binge eating and threshold/subthreshold bulimia nervosa than a widely used dietary restraint scale. Analyses confirmed that only a small subset of individuals (23%) above the median of the dietary restraint scale endorsed fasting in the past year, providing support for the notion that only a subset of individuals with elevated dietary restraint scores fast for weight control purposes. In addition, fasting showed significant relations with risk for onset in all eight models, whereas dietary restraint showed significant relations with risk for onset in only two of the eight models.

Furthermore, fasting was a stronger predictor of recurrent binge eating and threshold/subthreshold bulimia nervosa onset than the dietary restraint scale for six of the eight pairs of models (effects were small to large in magnitude). Similar findings emerged for the continuous and dichotomous predictors and for the models examining predictive effects over 1-year and 5-year periods. However, the 95% confidence intervals suggested that the predictive effect for the fasting variable was significantly larger than the predictive effect for dietary restraint for only two of the eight pairs of models. For example, the confidence intervals indicated that the predictive effect for the dichotomous fasting variable was significantly larger than the predictive effect for dichotomous dietary restraint variable in the models predicting bulimic pathology onset over the 5-year follow-up. Collectively, results provide preliminary support for the hypothesis that extreme fasting for weight control, practed by only a small subset of self-reported dieters, is a more potent risk factor for subsequent bulimic pathology than elevated

scores on dietary restraint scales. These findings converge with previous evidence that unhealthy weight control behaviors, which included fasting, use of food substitutes, meal skipping, cigarette smoking, diet pill use, vomiting, laxative/diuretic use, showed stronger relations to future increases in binge eating frequency relative to self-reported weight-loss dieting (Neumark-Sztainer et al., 2006).

Experiments have found that acute periods of marked caloric restriction increase the reinforcing value of food (Epstein, Truesdale, Wojcik, Paluch, & Raynor, 2003), which may explain why fasting increases the risk for binge eating. Acute periods of marked caloric restriction have also been shown to deplete tryptophan, an amino acid precursor of serotonin (Cowen, Clifford, Walsh, Williams, & Fairburn, 1996), which may increase the likelihood of binge eating high-carbohydrate food to restore tryptophan levels. Findings also converge with evidence that retrospectively reported weight suppression increases risk for the persistence of bulimia nervosa among those in treatment for this eating disorder (Butryn, Lowe, Safter, & Agras, 2006). However, these interpretations are based on the assumption that during fasting episodes participants consume fewer calories than people not engaging in fasting, which has not been confirmed with objective data.

It is noteworthy that two of the four predictive effects for dietary restraint were significant in the models examining the 1-year follow-up period, but none of the predictive effect for dietary restraint were significant in the models examine the full 5-year follow-up period, which was longer than the follow-up used in past studies that implicated dietary restraint as a risk factor for bulimic pathology (e.g., Johnson & Wardle, 2005; Killen et al., 1996). This pattern of findings may imply that the effects of dietary restraint exert their effect over a more proximal time interval. In contrast, fasting exerted predictive effects over both the shorter and the longer follow-up period, providing further evidence that fasting is a more robust risk factor for bulimic pathology. *Post hoc* analyses tested identical models using T-2 lagged predictors; all of the fasting effects remained significant whereas all of the dieting effects were non-significant in these models, providing additional support for the suggestion that the predictive effects of dietary restraint operate over a shorter period. It might be argued that the moderate 1-year temporal stability for the dietary restraint measure in the present study explains the null effects for this variable in the logistic regression models, however, the fasting variable showed even lower temporal stability, yet still exhibited consistent significant predictive effects.

We did not treat fasting as a compensatory behavior that would count toward a diagnosis of threshold/subthreshold bulimia nervosa and were careful to ensure that fasting predicted future onset of binge eating and bulimic pathology (e.g., by not examining any predictive effects of fasting for individuals how had already shown onset of binge eating or bulimic pathology) because we wanted to ensure that the effects were truly prospective. However, because fasting is a symptom of bulimic pathology, it could be argued that fasting is simply the first facet of this eating disorder to emerge developmentally. However, the fact that 87% of participants who reported fasting did not go on to develop threshold/subthreshold bulimic pathology over the following 5-years seems to argue against this alternative interpretation. Nonetheless, it will be important for future studies to address this possible interpretation in independent studies.

Another alternative interpretation that is worth considering is that some third variable is responsible for the predictive effects of dietary restraint or fasting. In support of this alternative explanation, dietary restraint predicted onset of bulimic pathology in a univariate model, but this effect became non-significant when other predictors were entered into the model (Killen et al., 1996). Unfortunately, it is impossible to rule out the possibility that any prospective effect observed in a longitudinal study is due to some unmeasured third variable. Even if potential third variables are measured, there is no way to determine which is responsible for a particular predictive effect because prospective studies simply do not permit firm inferences

regarding causality. For these reasons, it would be useful for future studies to experimentally manipulate (reduce) fasting behavior to determine whether it results in a reduction in bulimic pathology.

It is important to consider the limitations of this study when interpreting the findings. First, most of the data were provided by the participants, which increases the risk that reporter-bias inflated the magnitude of the relations. Second, there is limited evidence for the reliability and validity of our measure of fasting behaviors. Most importantly, we have no evidence that participants consume fewer calories during fasting episodes relative to participants who do not report fasting. Third, it is always possible that some third variable explains any relations observed in a prospective study, so these results should not be interpreted as establishing a causal relation. Future studies should strive to address these limitations.

In conclusion, the present results suggest that fasting for weight control purposes is a more potent and consistent predictor of risk for future onset of binge eating and bulimic pathology, rather than less severe dieting behaviors practiced by individuals with elevated scores on dietary restraint scales. An important direction for future research will be to conduct rigorously controlled experiments that reduce fasting among at-risk samples to determine whether this reduces risk for current and future onset of binge eating and bulimic symptoms. Such experiments are vital because they are much less vulnerable to third-variable alternative explanations than prospective etiologic studies.

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Table 1

Relations of Fasting and Dietary Restraint to Risk for Future Onset of Recurrent Binge Eating and Bulimic Pathology Over the 5-year Follow-up.

	Risk for onset of recurrent binge eating		
T1 Predictor Models	**Beta (95% CI)**	**Odds ratio**	***p*-value**
Continuous fasting variable	.31 (.04 – .58)	1.37	.021*
Continuous dietary restraint scale	.17 (−.16 – .50)	0.08	.303
Dichotomous fasting variable	.85 (.01 – 1.69)	2.35	.046*
Dichotomous dietary restraint scale	.45 (−.23 – 1.13)	1.56	.188
T-1 Propotional Hazards Models	Beta (95% CI)	Odds ratio	*p*-value
Continuous fasting variable	0.25 (0.01 – 0.48)	1.28	.039*
Continuous dietary restraint scale	0.37 (0.06 – 0.67)	1.44	.018*
Dichotomous fasting variable	0.96 (0.09 – 1.84)	2.62	.031*
Dichotomous dietary restraint scale	0.52 (−0.12 –1.17)	1.69	.112
	Risk for osnet of bulimic pathology onset		
T1 Predictor Models	Beta (95% CI)	Odds ratio	*p*-value
Continuous fasting variable	.51 (.20 – .82)	1.66	<.001***
Continuous dietary restraint scale	.15 (−.29 – .59)	1.16	.490
Dichotomous fasting variable	1.33 (.37 – 2.29)	3.80	.005**
Dichotomous dietary restraint scale	.28 (−.57 – 1.14)	1.32	.518
T-1 Propotional Hazards Models	Beta (95% CI)	Hazard ratio	*p*-value
Continuous fasting variable	0.31 (0.03– 0.58)	1.36	.030*
Continuous dietary restraint scale	0.45 (0.05– 0.84)	1.56	.028*
Dichotomous fasting variable	1.10 (0.01– 2.19)	3.01	.047*
Dichotomous dietary restraint scale	0.38 (−0.46 – 1.21)	1.46	.381

Note: CI = Confidence Interval.

* $p < .05$,

** $p < .01$,

*** $p < .001$