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Relations Between Dietary Restraint, Depressive Symptoms, and Binge Eating: A Longitudinal Study

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ABSTRACT

Objective: Temporal relations between dietary restraint, depressive symptoms, and binge eating were tested by means of three competing models positing that (1) dietary restraint and depressive symptoms predict future increases in binge eating, (2) binge eating predicts future increases in dietary restraint and depressive symptoms, and (3) binge eating is reciprocally related to these two factors.

Method: Longitudinal data from a community sample of Dutch females ($N = 143$; M age = 19.6) was used to test these relations while controlling for initial levels of these factors.

Results: Dietary restraint did not predict future increases in binge eating, nor

did binge eating predict future increases in dietary restraint. Depressive symptoms predicted future increases in binge eating, but binge eating did not predict future increases in depressive symptoms.

Conclusion: Although this study had limited statistical power, the pattern of relations and effect sizes suggest that depressive symptoms, but not dietary restraint, increase risk of binge eating for late adolescent females. © 2006 by Wiley Periodicals, Inc.

Keywords: dietary restraint; depression; binge eating; eating disorders

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Introduction

Binge eating is a prevalent eating disorder symptom, is associated with functional impairment and comorbid psychopathology, and increases the risk of onset and maintenance of obesity and a poor response to weight loss treatments.^{1–5} Accordingly, considerable research attention has focused on identifying risk factors for this key eating disorder symptom. Two risk factors that have received the greatest attention have been dietary restraint and negative affect.^{6–8}

Dietary Restraint and Binge Eating

According to the restraint theory,⁹ dieting entails a shift from a reliance on physiological control to cognitive control over eating behaviors, which

leaves dieters vulnerable to disinhibited eating when these cognitive processes are disrupted.¹⁰ In addition, the chronic hunger experienced by dieters theoretically increases the risk of binge eating.¹⁰ Violation of strict dietary rules may also result in the temporary abandonment of dietary restriction because of the abstinence violation effect.¹¹ Furthermore, dieting might result in depletion of tryptophan, a precursor of serotonin, which increases the likelihood of binge eating high-carbohydrate food to restore tryptophan levels.¹²

Consistent with the restraint theory, several prospective studies have found that self-reported dieting predicts future onset of binge eating among asymptomatic individuals.^{13–15} Furthermore, dieting predicted subsequent increase in bulimic pathology.^{16–19}

The results of other studies, however, have not provided support for the proposition that dietary restraint is positively related to binge eating. For example, certain studies did not demonstrate a significant relation between initial dieting and future increases in bulimic pathology.^{19,20} In addition, there is empirical evidence that dieting is negatively associated with binge eating. Several experimental studies have found that random assignment to low-calorie diets, which result in documented weight loss, produced greater decreases in binge

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eating than observed in waitlist controls in normal weight, overweight, and obese females.^{21,24}

Two other relations between dietary restraint and binge eating have also been posited.¹⁹ First, people who binge eat might attempt to avoid consequent weight gain by dieting. Within this context, Stice¹⁹ found that bulimic symptoms predicted future increases in dieting. Furthermore, retrospective studies suggest that bingeing behavior often precedes dieting restraint.^{25–29} Second, it has been posited that dietary restraint and binge eating may be reciprocally related.¹⁹ However, in contrast with this assumption, Stice¹⁹ did not find reciprocal relations between dietary restraint and bulimic symptoms in adolescent females. To the best of our knowledge, this latter study has been the only one to investigate these two alternative relations between dietary restraint and binge eating using a single prospective data set.

In an effort to gain a better understanding of the developmental course of the relation between dietary restraint and binge eating, it is important to replicate all three associations in an independent sample. Accordingly, the present study tested the three possible relations between dietary restraint and binge eating using data from a community sample of Dutch females.

Negative Affect and Binge Eating

According to the affect-regulation model,^{30,31} binge eating provides transitory comfort and distraction from aversive emotions. In support of this theory, self-reported negative affect predicted onset of binge eating among asymptomatic adolescent girls^{13–15} and future increases in bulimic symptoms in young women.³² However, a study involving adult women did not find a significant association between self-reported negative affect and onset of binge eating.³³

The opposite relation has also been posited.¹⁹ Binge eating might foster depressive feelings, shame, and guilt. Consistent with this hypothesis, bulimic symptoms have been found to predict future increases in depressive symptoms³⁴ and onset of major depression in adolescent girls.³⁵ Second, it has also been suggested that these processes may be reciprocally related.¹⁹ That is, negative affect and binge eating are supposed to be reciprocally related over time. In an earlier study, these reciprocal relations were studied for bulimic behavior.¹⁹ However, no support for these reciprocal relations were found. It appears that the latter study is again the only study in which these two alternative explanations have been tested at the same time.

In order to gain more insight in the temporal relationships between negative affect and binge eating, the aim of the present study was to replicate the three possible relations. Because Stice et al.¹⁵ found that elevated depressive symptoms, but not anxiety and anger, predicted binge eating onset, the relation between depressive symptoms and binge eating was examined in the present study.

Method

Participants

Participants were female undergraduates who volunteered in exchange for course credit and money. A total of 143 females participated at Time 1. The mean age was 19.6 years (range 17 to 38 years) at Time 1. Nearly all participants were born in The Netherlands (95%). Participation rate at Time 2 was 88% ($N = 127$). Most nonresponses were due to new, unknown addresses.

Procedure

The study was presented as an investigation of the relations of appearance orientation and negative emotions with eating attitudes, eating behaviors, and awareness of physical and emotional signals. At Time 1, all the participants completed the questionnaires in a classroom setting. At Time 2, the questionnaires were mailed to them. Completed questionnaires were returned by mail. There was a 12-month lag between both measurement points. This time interval was selected because earlier studies have shown that in this time span bulimic symptoms can develop or change.^{19,15}

Measures

Dietary restraint was measured with the 10-item Restrained Eating subscale of the Dutch Eating Behavior Questionnaire (DEBQ; 36). Items are rated on 5-point Likert scales, ranging from 0, *never*, to 5, *always*. The DEBQ-R has been found to show inverse correlations with self-reported caloric intake, but shows weaker relations to objective measures of acute caloric intake.^{36,37} Cronbach alpha (α) coefficients were 0.96 on Time 1 and 0.95 on Time 2.

The 16-item Depression subscale of the revised version of the Hopkins Symptom Checklist (SCL-90; 38; Dutch version: 39) was used to assess depressive symptoms. The SCL-90 is a well-known, reliable measure of clinical symptoms. The items are rated on 5-point Likert scale, ranging from 1, *not at all*, to 5, *very much*. The Dutch version of the SCL-90 has found to discriminate between individuals diagnosed with major depression and those without.³⁹ In the present study, Cronbach α coefficients were 0.91 and 0.90 for Time 1 and Time 2, respectively.

Binge eating was measured with the 7-item bulimia subscale of the revised Eating Disorder Inventory (EDI-

TABLE 1. Means, standard deviations, and bivariate correlations between the variables ($N = 143$)

	1	2	3	4	5	6	7	<i>M</i>	<i>SD</i>
1. Restrained Eating T1	—							2.67	0.94
2. Depression T1	0.29**	—						1.78	0.63
3. BMI T1	0.24*	−0.03	—					20.92	2.40
4. Binge Eating T1	0.48**	0.37**	0.33**	—				2.09	0.87
5. Restrained Eating T2	0.80**	0.30**	0.32**	0.45**	—			2.72	0.91
6. Depression T2	0.24*	0.46**	0.02	0.26*	0.31**	—		1.72	0.58
7. Binge Eating T2	0.46**	0.41**	0.32**	0.81**	0.49**	0.38**	—	2.12	0.77

Note: T1 = measurement point 1; T2 = measurement point 2; 12 months after T1.

* $p < 0.01$,

** $p < 0.001$.

II; 40; Dutch version: 41). Items are rated on 6-point Likert scales, ranging from 1 *never*, to 6, *always*. The Dutch version of the EDI-II has been shown to discriminate between nonclinical females and eating disordered females.⁴² The Bulimia subscale generally provides coverage for binge eating, but not compensatory behaviors or weight and shape overvaluation. As a result, this scale does not map particularly well bulimia nervosa as outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR⁴³). Therefore, we decided it was best to acknowledge that this scale primarily measures binge eating. As a result, the item referring to compensatory behavior was left out of the analyses. Cronbach's α coefficients for the subscale consisting of only binge-related items were 0.89 and 0.85 for Time 1 and Time 2, respectively. The original Bulimia scale and the subscale Binge Eating were highly intercorrelated: $r = 0.99$ for Time 1 as well as for Time 2.

Results

Preliminary Analyses

Participants who provided data on both time points ($N = 127$) did not differ significantly from those who only provided baseline data on Restrained Eating, Depression, or Binge Eating at Time 1 ($N = 143$) (all p values of >0.05). As can be seen in **Table 1**, mean scores were relatively stable over time.

Test of Competing Models

The correlations between the initial levels of Restrained Eating, Depression, and Binge Eating on Time 1 with the corresponding levels at Time 2 indicate relative stability of these factors (see **Table 1**). Furthermore, the correlations between the factors indicate that they may be related over time. However, a predictor must account for variance in a criterion over and above the temporal stability of that criterion.⁴⁴ Therefore, the prospective relations between the factors were examined while statistically controlling for the initial levels of these factors. The prospective relations were tested by

means of structural equation modeling,⁴⁵ using the AMOS 5.0 statistical package.⁴⁶ Full information maximum-likelihood estimation (FIML) was used to handle missing data because FIML produces more accurate and efficient parameter estimates than do listwise deletion or alternative imputation approaches and maximizes statistical power.⁴⁷ Furthermore, backward elimination of nonsignificant parameters⁴⁸ was used to obtain the final models. Backward elimination allows for the examination of all predictors in the equation and avoids certain types of specification errors.⁴⁸ Several goodness-of-fit measures were applied. First, the Chi square (χ^2), that has to be nonsignificant in order to accept a model. This means that the model provides an acceptable fit to the data, if it has a probability level of $p > 0.05$. Second, the Root Mean Square Error of Approximation (RMSEA), which should be less than 0.05 to indicate close fit.⁴⁹ Finally, we relied on the Comparative Fit Index (CFI) and the Tucker-Lewis Index (TLI). Both should be ≥ 0.95 .⁵⁰

Relations Between Dietary Restraint, Depressive Symptoms, and Binge Eating

Three alternative models for the relations between restraint, depressive symptoms and binge eating were compared with a baseline stability model. This stability model included only the paths from the Time 1 exogenous (independent) variables to the corresponding Time 2 endogenous (dependent) variables (see **Figure 1**). Furthermore, the error terms of the endogenous (dependent) variables could be correlated. The introduction of the error terms implies that not all of the associations between the variables are completely explained by their dependence on the observed exogenous variables. The model indicates that initial levels of Restrained Eating, Depression, and Binge Eating are markedly related to the outcome levels of these factors at Time 2. The model fitted the data reasonably well (see **Table 2**). The three models representing our hypotheses were compared with this baseline model.

FIGURE 1. Baseline model for the links between Restrained Eating and Binge Eating and between Depression and Binge Eating. *** $p < 0.001$.

Model 1 represents the restraint model and the affect-regulation model. In this model, paths from Time 1 Restrained Eating and Time 1 Depression to Time 2 Binge Eating were specified (see **Figure 2**). The path from Time 1 Restrained Eating to Time 2 Binge Eating was not significant. As a result, no support was found for the relation between Restrained Eating and future increases in Binge Eating. As expected, Time 1 Depression predicted increases in Binge Eating from Time 1 to Time 2 ($r = 0.19$). As the path coefficient of Restrained Eating was nonsignificant, this coefficient was omitted from the model. This restricted Model 1 fit the data well (see **Table 2**). Furthermore, from the significant chi-square change, it can be concluded that the Model 1 fit the data significantly better than the baseline model.

In Model 2, paths from Time 1 Binge Eating to Time 2 Restrained Eating and Time 2 Depression were tested (see **Figure 3**). It was found that these paths were not significant. Thus, these results did not support the competing hypothesis that binge eating predicts future increases in dietary restraint and depressive symptoms over and beyond stability. The nonsignificant path coefficients were omitted from the model, resulting in a restricted Model 2 that fit the data sufficiently (see **Table 2**). However, the nonsignificant chi-square change indicates that Model 2 did not fit the data significantly better than the baseline model.

Model 3 tested reciprocal relations over time between Restrained Eating and Binge Eating and also between Depression and Binge Eating (see **Figure 4**). The results indicate that Restrained Eating and Binge Eating were not reciprocally related over time. That is, Time 1 Restrained Eating did not predict future increases in Binge Eating and Time 1

TABLE 2. Test of competing models for binge eating ($N = 143$)

Model	χ^2	df	p	RMSEA	CFI	TLI	χ^2 Change ^a
Baseline model	8.910	4	0.06	0.09	1.00	1.00	—
Model 1	2.93	2	0.23	0.06	1.00	1.00	5.98*
Model 2	5.32	2	0.07	0.11	1.00	1.00	3.59
Model 3	3.97	3	0.26	0.05	1.00	1.00	4.94*

^a All Chi-square change values reflect a comparison of the model to the baseline model.

* $p < 0.05$.

Binge Eating did not predict future increases in Restrained Eating over and beyond stability. Also, no reciprocal relations were found between Depression and Binge Eating. Time 1 Depression predicted future increases in Binge Eating, but Time 1 Binge Eating did not predict future increases in Depression. The nonsignificant path coefficients were omitted from the model. The fit indices indicated that this restricted Model 3 fit the data well (see **Table 2**). Furthermore, Model 3 fit the data significantly better than the baseline model.^a

Effect Sizes and Power Computations

Because the sample size ($N = 143$) was only moderately large, one could ask whether the nonsignificance of several of the regression coefficients could be due to a relatively low power. With an N value of 143, the power for detecting a medium effect size

^aIt should be noted that the pattern of effects was identical (i.e., all significant effects remained significant and all nonsignificant effects remained nonsignificant) when two separate series of models were used to examine the relation of binge eating to dietary restraint and to depression. It should also be noted that the same pattern of effects emerged when the relations for EDI-II Bulimia subscale⁴⁰ were examined, with one exception: T1 Bulimia predicted T2 Depression in Model 3 ($\beta = .22$, $p < 0.05$).

FIGURE 2. Associations between Time 1 Restrained Eating and Time 2 Binge Eating and between Time 1 Depressive Symptoms and Time 2 Binge Eating. * $p < 0.05$, *** $p < 0.001$.

FIGURE 3. Associations between Time 1 Binge Eating and Time 2 Restrained Eating and between Time 1 Binge Eating and Time 2 Depressive Symptoms. *** $p < 0.001$.

($r = 0.30$) is high: 0.85 at $\alpha = 0.05$.⁵¹ Specifically, in the present study, the effect size found for dieting on future increases in bulimic symptoms as measured by the full bulimia subscale of the EDI-II⁴⁰ was $r = 0.17$. This is in the range of the effect sizes found in previous prospective studies testing this effect ($r = 0.15$).⁵² The effect size for dieting on future increases in binge eating in the present study was also $r = 0.17$. With an N value of 143, the power for detecting an effect size of $r = 0.15$ was moderate: 0.50.

Conclusion

Relations Between Dietary restraint and Binge Eating

Dietary restraint did not predict future binge eating. The lack of a relation between initial dietary

restraint and future binge eating is inconsistent with that found in an earlier study in adolescent girls,¹⁵ but it is consistent with another study that has found that initial dietary restraint scores did not predict future increases in bulimic symptoms.¹⁹ A possible explanation might be the differences in follow-up period and measurement points. In the present study a follow-up period of 12 months with two measurement points was used. Stice et al.¹⁵ had a longer follow-up period (20 months) with three measurement points, which increased the power to detect changes in binge eating. In line with this interpretation, the effect size from Stice et al.¹⁵ was $r = 0.25$ versus $r = 0.17$ in the present study. Furthermore, the effect size found in the present study is very similar for the average effect size from a meta-analytic review,⁵² which suggests that this effect size is rela-

FIGURE 4. Reciprocal relationships between Restrained Eating and Binge Eating and between Depressive Symptoms and Binge Eating over time. * $p < 0.05$, *** $p < 0.001$.

tively small and likely to be inconsistently observed, particularly in smaller studies. Another possible explanation might be the difference in sample characteristics, in particular age. Stice et al.¹⁵ found a prospective effect in a sample of young adolescents, whereas we failed to find an effect in female college students. It might be possible that dietary restraint predicts binge eating in middle adolescence, but not in late adolescence. Also in line with this interpretation, the high stability coefficient for binge eating (stability coefficient varying from 0.73 to 0.80), which made it difficult to detect predictors of change in binge eating, suggests that this eating behavior is relatively stable by late adolescence.

Findings of the present study also indicate that initial binge eating did not predict future dieting. This finding is inconsistent with the outcomes of an earlier study,¹⁹ showing that bulimic behaviors predicted future dietary restraint while controlling for initial restraint scores. It might be argued that the discrepant findings emerged because the present study examined binge eating, whereas Stice¹⁹ examined the broader construct of bulimic symptoms. It might be that bulimic pathology, but not specifically binge eating, is related to future dietary restraint. However, in the present study also no support was found for an effect of the EDI-II bulimia subscale⁴⁰ on future increases in dietary restraint. Another explanation might be that the higher mean age of the present sample influenced the relation between binge eating and dietary restraint. That is, binge eating predicts future dietary restraint in middle adolescence, but not in late adolescence. Finally, the effect size from bulimia to future dietary restraint observed in Stice¹⁹ was $r = 0.17$, whereas we found an effect size of $r = 0.10$.

These findings indicate that the effect size is relatively small and, therefore, might be inconsistently observed in smaller studies.

Relations Between Depressive Symptoms and Binge Eating

Initial depressive symptoms predicted future binge eating, which replicates effects observed in earlier prospective studies.^{13,53} This significant relation provides support for the affect-regulation model,^{30,31} which posits that people may binge eat to provide comfort and distraction from adverse emotions.

Although earlier studies found that bulimic symptoms predict depressive symptoms and onset of depression,^{35,34} we did not find a significant relationship between initial binge eating and future depressive symptoms. A possible explanation is that not so much binge eating, but, more generally, bulimic pathology is related to future depression. In support of this assumption, in the present study it was found that in Model 3 the EDI-II subscale Bulimia⁴⁰ predicted Time 2 Depression (see footnote).

In general, the results of the present study do not support the expectations regarding relations between dietary restraint and binge eating over time. The findings also do not provide evidence for the hypothesis that binge eating is related to future depressive symptoms. However, the results do provide additional evidence that binge eating might be rooted in efforts to regulate negative affect. Finally, the findings of the present study show that initial levels of dietary restraint, depressive symptoms, and binge eating were strongly associated with respective levels of these same outcomes 12 months later, indicating that these factors are relatively stable in women during young adulthood.

Limitations and Directions for Future Research

It is important to consider the limitations of this study when interpreting the findings. First, although the prospective design provides evidence regarding the effects of the factors, causal order cannot be definitely determined as other variables, not included in the study, can account for the observed relations. Second, the results found in this study should be interpreted with caution, as the sample size is relatively small. Third, the current sample was relatively homogeneous regarding age and education. Thus, these results may not be generalized to early adolescent and adult populations, or to male and clinical eating-disordered samples. Fourth, the study relied on self-report measures. Therefore, results may be influenced by several inaccuracies, such as social desirability biases.

Generally, the results of the present study do not provide support for the assumption that dietary restraint predicts binge eating. It might be possible that other variables, such as age, play an important role in the detection of the effect of dietary restraint on future binge eating. Future research should examine whether dietary restraint predicts binge eating only within various developmental periods, such as early and middle adolescence. Furthermore, because dietary restraint reduced binge eating in several randomized trials,^{21–24} more research is needed to investigate the effects of various forms of dieting on binge eating. It might be possible that dietary restraint has different effects on binge eating when dieting involves flexible versus rigid control of eating behaviors^{54,55} or whether it involves a temporary weight loss diet versus chronic restraint in eating behavior.⁵⁶

Moreover, the results of the present study imply that depressive symptoms may play an important role in the development of binge eating. This finding points to the need for more research into risk factors that predict increases in depressive symptoms and into the nature of the relation between depressive symptoms and binge eating. For example, it is possible that the relation between depression and binge eating is mediated by a number of factors, such as lack of interoceptive awareness.⁵⁶ Finally, there may be other important risk factors for binge eating that remain to be discovered, such as personality variables (i.e., impulsivity^{57–60}), and biological processes (i.e., tryptophan¹²). Future studies should begin to explore how depression might work in conjunction with these other potential risk factors.

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