Published in final edited form as:
Int J Eat Disord. 2007 January ; 40(1): 27–36.

An Experimental Test of the Affect-Regulation Theory of Bulimic Symptoms and Substance Use: A Randomized Trial

Emily Burton, M.A.[1], **Eric Stice, Ph.D.**[1], **Sarah Kate Bearman, Ph.D.**[1], and **Paul Rohde, Ph.D.**[2]

1*Department of Psychology, University of Texas at Austin*

2*Oregon Research Institute, Eugene, Oregon*

Abstract

Objective: Conduct a randomized trial to test whether a cognitive behavioral intervention designed to decrease depressive symptoms produces subsequent decreases in bulimic and substance use symptoms. **Methods:** Female participants (N=145) with elevated depressive symptoms were randomly assigned to a 4-session depression intervention or a measurement-only condition and assessed through 6-month follow-up. **Results:** Relative to control participants, intervention participants showed decreases in depressive symptoms. Intervention participants also showed significantly greater reductions in bulimic symptoms, but not substance use, and change in depressive symptoms mediated this effect for bulimic symptoms. **Discussion:** Results provide experimental support for the theory that affect disturbances contribute to bulimic pathology, but do not support the affect regulation theory of substance use.

Keywords

affect-regulation; depression; bulimia; substance use; randomized trial

---

An Experimental Test of the Affect-Regulation Theory of Bulimic Symptoms and Substance Use: A Randomized Trial

Numerous theorists have posited that negative affect increases risk for certain addictive behaviors, including binge eating and bulimic pathology, as well as substance use. Specifically, the negative affect model of bulimic pathology proposes that individuals may binge eat because they believe it provides distraction and comfort from painful negative emotions (1-2). This theory also suggests that individuals might use radical compensatory behaviors, such as vomiting or laxative abuse, to reduce anxiety about impending weight gain consequent to overeating or because they believe that purging provides an emotional catharsis. In support of this theory, depressive symptoms and negative affect have been found to predict future increases in bulimic symptoms (3-4), as well as onset of binge eating (5), bulimic pathology (6-8), and general eating pathology (9). Although several prospective studies have reported null findings for these relations (e.g., 1,10-11), these studies were more likely to have used smaller samples and focused on general eating pathology outcomes.

Theorists have also proposed that depression is a risk factor for the development of substance use and abuse (12-15). Theoretically, depressed individuals may consume psychoactive substances to improve their mood or as an escape from adverse emotions. The reinforcement

Address correspondence to Dr. Eric Stice at Oregon Research Institute, 1715 Franklin Blvd., Eugene, Oregon, 97403. Email: estice@ori.org..

received may lead to elevated substance use rates and subsequently the use-related negative consequences that characterize substance abuse. Because mood disturbances increase markedly for girls during adolescence (16), depressive symptoms might be a particularly important predictor of substance use for this population. Depressive symptoms and negative affect have been found to prospectively predict future increases in substance use (17-21), though null findings have been reported (22-24). While issues of sampling variability may contribute to these inconsistent findings, no obvious factors, such as sample size, outcome measures utilized, assessment intervals, or length of follow-up, appear to readily shed light on these discrepancies. Thus, it is possible that this effect is simply smaller, and therefore only inconsistently observed.

One limitation of this body of research is that all of these prospective effects may be due to omitted third variables. Although prospective studies are an improvement over cross-sectional designs because they can provide evidence of temporal precedence, it is always possible that some confounding variable gives rise to both the putative risk factor and future increases in the outcome variable (25). Randomized trials afford a unique opportunity to attempt to experimentally confirm findings from prospective risk factor studies and such trials provide powerful tests of theories, given their relative immunity to the effects of third-variables (26-28). Further, a randomized trial is one of the few experimental paradigms that could potentially result in long-term decreases in depressive symptoms, providing an advantage over laboratory experiments that manipulate acute mood. In terms of affect regulation, if individuals engage in bulimic behaviors and substance use to reduce negative affect, it follows that an intervention that reduces depressive symptoms should produce consequent decreases in bulimic symptoms and substance use. To our knowledge, these relations have not been examined in a randomized prevention trial that manipulated longer-term depressive symptoms. If such a randomized trial generates findings that converge with those from prospective risk factor studies, greater confidence can be placed in the hypothesized relations. Finally, evidence that reduction of depressive symptoms decreases bulimic symptoms and substance use would be beneficial for informing prevention efforts, because it would suggest that it might be possible for a single intervention to reduce all three of these pernicious psychiatric disturbances. Prevention programs that target what might be referred to as *linchpin* disturbances that promote other disturbances may effectively reduce risk for multiple problems, and therefore would be more desirable from a public health perspective than programs that impact only one disturbance.

To achieve this end, we developed a brief, 4-session cognitive-behavioral therapy (CBT) intervention to reduce depressive symptoms because prevention and treatment interventions for adolescent depression that focus on negative cognitions appear to be highly effective interventions (29-31). The resulting intervention briefly introduces the cognitive-behavioral model of depression, helps participants identify negative cognitions and precipitating circumstances, and teaches them to generate positive thoughts that can be used to replace negative cognitions. Furthermore, because treatments that focus on instigating active participation in reinforcing activities have been found to be efficacious for alleviating depression in adult samples (32-33), our intervention also encourages participants to re-engage in activities that used to give them pleasure (e.g., playing a favorite sport) and to partake in a wider variety of pleasurable activities to improve mood.

We based our program on the Clarke et al. (30) intervention for high-risk dysthymic adolescents because it appeared to be the most promising extant program. Clarke et al. (30) randomly assigned 150 high-risk adolescents to a 15-session CBT intervention or an assessment-only usual care control condition. Intervention participants showed significantly greater reductions in depressive symptoms from pre- to post-treatment, but this effect was no longer significant at 12-month follow-up. Intervention participants also showed significantly lower rates of onset

of major depression over the 1-year follow-up relative to controls (15% versus 26% respectively). A follow-up trial involving 94 adolescents with subdiagnostic depression who were offspring of depressed parents also found that intervention participants showed significantly larger decreases in depressive symptoms from pre- to post-treatment and lower risk for subsequent onset of major depression (34). Though this program is promising, the long duration of the intervention (15 sessions) renders it challenging to implement. Thus, we developed and evaluated a brief CBT intervention believing that it would be more appealing to adolescents and easier to implement.

We drew upon the content of the Clarke program as a starting point, as well as our experience with the design of eating disorder prevention programs (35). Several principles guided the development of our intervention. Didactic presentation was minimized because psychoeducational interventions are less effective than interventions that actively engage participants (35). Numerous in-session exercises were used that require youth to apply the skills taught in the intervention. We used homework to reinforce the skills taught in the sessions and help participants learn how to regularly apply these skills. More generally, we used motivational enhancement exercises to maximize motivation to use the new skills, strategic self-presentation to facilitate internalization of key principles, behavioral techniques to reinforce use of the new skills, and group activities to foster feelings of social support and group cohesion.

Methods

Participants

Participants were 145 female students recruited from two high schools and one college who ranged in age from 14 to 23 (M = 18.6, SD = 1.8) at baseline (T1). We focus solely on young women for this trial because bulimic symptoms are much more common in women than in men (36). The sample was composed of 18% Asians, 6% Blacks, 52% Caucasians, 19% Hispanics, 1% Native Americans, and 4% who specified *other* or mixed racial heritage. Educational attainment of parents, a proxy for socioeconomic status, in our sample (22% high school graduate or less; 22% some college; 35% college graduate; 21% graduate degree) was generally similar to regional census data (34% high school graduate or less; 25% some college; 26% college graduate; 15% graduate degree).

Procedures

Participants were recruited using mass mailings (high school students) and emails (college students), handbills distributed after classes, and posted fliers that invited students between the ages of 14 and 23 suffering from feelings of depression to participate in a trial of an intervention designed to improve mood. Participants interested in the intervention who reported elevated depressive symptoms on a screening measure (Center for Epidemiologic Studies-Depression [CESD; (37)] scores $\geq$ 21) were randomly assigned, within blocks created by school, to either the brief CBT intervention condition or a waitlist control condition.

All participants who called for screening were provided with a referral phone number for free or low-cost treatment. Study consent forms encouraged participants and their parents to seek professional help if they had serious concerns about depression and indicated that study coordinators would help them access treatment. Following randomization, all qualifying participants in both conditions were informed that they should seek treatment if they experienced a depressive episode, were provided with a list of referral numbers, and were told that study coordinators could provide additional referrals if necessary. Further, all questionnaires were examined to assess risk to self. When participants endorsed suicidal ideation, these responses were followed up with a phone call to the participant. In all cases

where participants were contacted regarding their responses, we contracted for safety, encouraged them to seek treatment provided referral information, and documented the conversation. An emergency response plan was developed for participants who reported suicidal ideation or plans, but fortunately we never needed to implement these procedures.

The CBT depression intervention consisted of four weekly 1-hour sessions. Intervention groups were composed of 6-10 participants. Sessions were facilitated by a female clinical graduate student and co-facilitated by a female undergraduate. If a participant missed a session, we conducted a brief (10-15 minute) individual session with the participant prior to their next session in order to review missed material. Participants completed a 5-page survey at baseline, termination, 1-month follow-up, and 6-month follow-up that inquired about participants' feelings and behaviors over the past week and participants in the two conditions completed identical measures at parallel times. Participants provided written informed consent and parental consent was secured for minors. Participants were compensated $40 for completing all surveys.

CBT Depression Prevention Intervention

Session 1. First, the purpose of the group (overcoming sadness and preventing future sadness) is explained and the agenda for the session is presented. Second, a get-acquainted activity is used to build group rapport. Next, the emotional and social costs of depression are discussed as a means of motivational enhancement. Participants are then introduced to the CAB (Consequence, Activating event, Beliefs) method for identifying and reducing negative or irrational thoughts. This method involves noticing the Consequence (negative affect), identifying the Activating event (trigger), and determining the Beliefs that led from the activating event to the consequence. This model is used to demonstrate the impact of thinking on emotions, and to help participants identify automatic negative thoughts. The CAB model is supplemented with a discussion on the types of situations in which negative thoughts typically occur. Participants check off their own frequent negative thoughts from a checklist of common negative thoughts and share with group members one of their most frequent negative thoughts. This activity is followed by an in-depth discussion of activating events and how to recognize them. Finally, participants are asked to keep track of negative thoughts and activating events over the next week on a CAB tracking form. As a method of behavioral activation, participants are also asked to generate a list of pleasurable activities that they used to enjoy doing and to engage in one of these activities before the next session.

Session 2. This session begins with a review of the costs of negative thinking. The homework assignments are then reviewed. Participants share one of the activating events from their CAB tracking forms that resulted in negative feelings during the past week. Next, there is a discussion about increasing positive thinking. Participants then pair up and take a few minutes to get to know one another. Each participant shares aloud two positive things about their partner that they learned during the conversation and two positive things about themselves. The participant's partner does the same, but must disclose two novel positive things about themselves and their partner. This exercise illustrates how positive thinking about oneself and others improves mood. Participants are introduced to the concept of depressionogenic cognitions and instructed on challenging those cognitions; specifically, they discuss methods for countering negative thoughts with positive ones and how to identify and challenge irrational thinking. Each participant shares one of the negative thoughts listed on her CAB tracking form from the past week and the other participants work together to challenge that negative thought. Members of the group are then asked to write a contract aimed at reducing negative thinking and to select a reward for meeting their goal of reducing negative cognitions (e.g., take a bubble bath, listen to a favorite CD). For homework, participants attempt to identify negative thoughts *in vivo* once per day and to replace them with positive thoughts. Participants reward themselves

as per their contract as a positive reinforcer for reducing negative thoughts. Participants also continue filling out their CAB tracking forms and partake in another pleasurable activity from the list they generated.

Session 3. This session begins with a discussion of the pleasurable activities that participants have been engaging in outside of the group and the impact of those activities on their mood. A review of the CAB method follows. Next, participants use their CAB tracking forms to apply the CAB method. Each participant shares one negative thought from their list, its activating event, their positive counter-thought, and the reward they earned based on their contract. If participants have difficulty with any of the CAB steps, group members help troubleshoot. For homework, participants to fill out their CAB tracking forms, using their contract and rewarding themselves for successful negative thought replacement, and engage in another pleasurable activity during the week.

Session 4. During the final session, participants check in with the group regarding their progress with the CAB method. A discussion follows during which an alternate technique to stop negative cognitions – scheduled worry time – is introduced to supplement the CAB method, if necessary. Participants are also introduced to the concept of using daily prompts to remind them to think positively and to continue using the CAB method. Next, the group discusses how to plan ahead for major life events that might result in negative thinking and depressed mood (depression prevention). Participants brainstorm how to avoid such situations (if possible) and ways to cope with them if they are unavoidable. Participants are provided with a prevention plan worksheet to complete in session, and are asked to generate both major events and daily hassles that will likely occur. Next, they develop a plan for how they will cope with such an event using cognitive restructuring, behavioral activation, and other strategies from the previous sessions.

Waitlist Control Condition

Participants in the control condition were told that it was necessary to observe the changes in the outcome variables among individuals that did not receive our intervention. However, these participants were allowed to seek usual-care in any treatment form (e.g., psychotherapy or psychotropic medication) and were offered the intervention when all assessments had been completed. Overall, 7% of the participants sought some form of treatment during the study period. Treatment seeking rates were not significantly different across conditions (χ^2 [N = 123] = 1.26, p = .315).

Measures

Depressive symptoms. The 21-item Beck Depression Inventory – I (BDI) (38-39) was used to measure depression symptoms. For each item, participants select among four alternative responses reflecting the increasing levels of symptom severity, where 0 = no symptom present and 3 = severe symptom presentation. Items were summed to form an overall symptom composite. The BDI has acceptable internal consistency (α = .73 to .95), reliability (test-retest r = .60 to .90), and convergent validity with clinician ratings of depressive symptoms (M r = .75) (38). The BDI had an α = .88 at T1.

Bulimic symptoms. The 12 diagnostic items assessing DSM-IV symptoms of bulimia nervosa from the Eating Disorder Examination-Questionnaire (EDE-Q)(40) were used to assess bulimic symptoms. The EDE-Q is derived directly from the Eating Disorder Examination interview (EDE)(41), a validated measure of eating pathology. The EDE-Q assesses the main behavioral (binge eating and purging) and cognitive (weight and shape overvaluation) features of bulimia. The items were summed to form an overall bulimic symptom composite. This scale

has been found to possess adequate internal consistency (α = .84), 3-week test-retest reliability (*r* = .89), and discriminant validity (4,40,42). This scale had an α = .83 at T1.

Substance use. Adolescent substance use was measured with items from the Monitoring the Future Study (43). Participants reported their frequency of consumption during the past month of beer/wine/wine coolers, hard liquor, cigarettes, marijuana, stimulants, downers, inhalants, and hallucinogens using 6-point response scales ranging from *never* to *3-7 times a week*. Participants also reported the typical number of drinks consumed on an average drinking occasion and the typical number of cigarettes smoked per day. Items were summed to form an overall substance use composite. Past research has found these items possess adequate internal consistency (α = .87), one-year test-retest reliability (*r* = .50), and predictive validity (24,43). This scale had an α = .81 at T1.

Results

Preliminary Analyses

Preliminary analyses indicated that participants assigned to the two conditions did not differ significantly on the BDI, bulimic symptoms, substance use, age, ethnicity, or parental education at baseline, suggesting that randomization succeeded in creating initially equivalent groups (*p*-values > .10). Preliminary multiple regression analyses also provided no evidence that participant age, ethnicity, or parental education moderated intervention effects on any of the outcome variables.

The 20 (14%) participants who dropped from the study before providing follow-up data (13% waitlist and 15% CBT condition) did not differ from those who provided complete data on any of these baseline variables, nor did attrition differ significantly across groups (*p*-values > .10), providing evidence that attrition did not systematically bias the results. Nonetheless, we used full information maximum likelihood estimation, based on expectation-maximization algorithm, to impute missing data points because this approach produces more accurate and efficient parameter estimates than listwise deletion or alternative imputation approaches such as last-observation-carried-forward or mean imputation (44). It should be noted that the pattern of effects was similar when the more common approach of listwise deletion was used (i.e., all significant effects remained significant and all non-significant effects remained non-significant). Participants who provided complete data attended all sessions and completed all homework assignments.

Preliminary analyses also investigated the test-retest reliability of the outcome measures in the control group over the initial one-month period, to examine the reliability of these measures in the current study. Results indicated that the BDI, bulimic symptoms, and substance use measures evidenced acceptable one-month test-retest reliability in the control group (*r*'s = .85, .95, and .98 respectively).

Intervention Effects for Depressive Symptoms

To confirm that our intervention reduced depressive symptoms, we conducted repeated measures ANOVA models for the BDI to test whether the changes in depressive symptoms were significantly greater for the CBT intervention condition than for the control condition. Separate models tested for effects from pretest to posttest, pretest to one-month follow-up, and pretest to 6-month follow-up to provide a comprehensive picture of how long the intervention effects persisted over time. We used 2-tailed tests throughout.

Time-by-condition interactions indicated that there were significantly greater reductions in depressive symptoms in the CBT condition than in the control condition from pretest to posttest (F [1/143] = 70.89, $p < .001$, 33.1% variance explained), pretest to 1-month follow-up (F

[1/143] = 13.14, $p < .001$, 8.4% variance explained) and pretest to 6-month follow-up (F [1/143] = 5.75, $p = .018$, 3.9% variance explained). Table 1 reports the cell means and standard deviations at each assessment, along with the results of the pairwise comparisons. Follow-up paired t-tests (Bonferroni corrected $\alpha = .016$) verified that the reductions in depressive symptoms in the CBT condition were statistically significant. Although the decreases from pretest to 1-month follow-up and from pretest to 6-month follow-up in the waitlist condition were statistically significant, the time-by-condition interactions indicated that the reductions were significantly larger in the CBT condition than in the control condition.

Intervention Effects for Bulimic Symptoms

Repeated measures ANOVA models also tested whether there were significantly greater decreases in bulimic symptoms in intervention participants relative to controls. As hypothesized, there were significantly greater decreases in bulimic symptoms from pretest to posttest (F [1/143] = 8.67, $p = .004$, 5.7% variance explained) and pretest to 1-month follow-up (F [1/143] = 5.93, $p = .016$, 4.0% variance explained) in the intervention condition relative to the control condition. However, these effects were non-significant at 6-month follow-up (F [1/143] = 0.76, $p = .384$, 0.5% variance explained). Table 1 reports the means and standard deviations at each assessment, along with the results of the pairwise comparisons. Follow-up paired t-tests (Bonferroni corrected $\alpha = .016$) verified that the reductions in bulimic symptoms in the CBT condition were statistically significant from pretest to posttest, pretest to 1-month follow-up, and pretest to 6-month follow-up, although the decrease from pretest to 6-month follow-up in the control group was also statistically significant.

To ensure that these effects did not emerge solely because the intervention impacted the cognitive symptoms of bulimia nervosa (overvaluation of weight and shape), we conducted *post hoc* analyses testing for intervention effects on the overall bulimic symptom composite when the two items assessing overvaluation of weight and shape were omitted. The pattern of findings remained unchanged: intervention participants showed significantly greater decreases in the behavioral symptoms of bulimia nervosa from pretest to posttest (F [1/143] = 6.45, $p = .012$, 4.3% variance explained) and pretest to 1-month follow-up (F [1/143] = 4.44, $p = .037$, 3.0% variance explained), but not from pretest to 6-month follow-up (F [1/143] = 0.24, $p = .622$, 0.2% variance explained).

We conducted additional follow-up analyses to test the hypothesis that change in depressive symptoms mediated the effects of the intervention on change in bulimic symptoms. These analyses focused on pretest to posttest changes because this is the interval during which the mediated effects would be strongest, given that this was when the participants received the intervention. Consistent with this reasoning, the intervention effects for both depressive symptoms and bulimic symptoms were the largest during the pretest to posttest interval. Extending Baron and Kenny's (45) criteria for mediation to prospective data from a randomized prevention trial, four conditions must be met to infer mediation. First, the independent variable (intervention condition) should predict change in the outcome. In support, a regression model confirmed that treatment condition predicted pretest to posttest change scores (posttest – pretest) for bulimic symptoms ($\beta = -.24$, B = −2.07, 95% CI = −3.46 – −0.67, $p < .004$). Second, the independent variable should predict change in the mediator. In support, a regression model confirmed that treatment condition predicted pretest to posttest depressive symptom change scores ($\beta = -.58$, B = −9.19, 95% CI = −11.34 – −7.05, $p < .001$). Third, change in mediator should correlate with change in the outcome. In support, pretest to posttest depressive symptom change scores were significantly correlated with pretest to posttest bulimic symptom change scores ($r = .29$, $p < .01$). Fourth, the effect of the independent variable on change in the dependent variable should become non-significant when the effects of change in the mediator is statistically controlled for full mediation. A multiple regression model indicated that

treatment condition was no longer significantly related to bulimic symptom change scores when the effects of depressive symptoms change scores was statistically controlled (β = −.11, B = −0.92, 95% CI = −2.60 – 0.75, p = .275). Results are consistent with the suggestion that change in depressive symptoms mediated the effect of the intervention on change in bulimic symptoms.

Intervention Effects for Substance Use

Repeated measures ANOVA models tested whether there were significantly greater decreases in substance use in intervention participants than in controls. There were no significant differences in change in substance use from pretest to posttest (F [1/143] = 0.36, p = .548, 0.0% variance explained), pretest to 1-month follow-up (F [1/143] = 0.21, p = .647, 0.0% variance explained) or pretest to 6-month follow-up (F [1/143] = 0.13, p = .714, 0.0% variance explained) across conditions. Means and standard deviations for the two conditions at each of the assessments are provided in Table 1.

Discussion

The aims of this trial were to test negative affect theories that assert that depressive symptoms increase the risk for bulimic symptoms and substance use (2,14). We used a randomized trial in an attempt to provide an experimental test of these theories because there is growing recognition that this is a rigorous method of testing hypotheses (26-28,46) as it is always possible that the prospective effects observed in longitudinal studies are due to some unmeasured third-variable. However, controlled trials greatly reduce this possibility because random assignment dramatically increases the likelihood that third-variables are uncorrelated with the experimental manipulation by equally distributing them across conditions. Randomized trials are also uniquely suited to this objective because they provide one of the few experimental paradigms that could manipulate depressive symptoms for a prolonged period of time. Although the use of randomized intervention trials for theory testing has certain limitations, including concerns regarding the ecological validity of the findings for etiologic processes, we think that there is great value in utilizing both prospective and experimental designs to test hypotheses. If the conclusions from both types of studies agree, much greater confidence can be placed in the theory, and we consider this a critical direction for future research in this field.

As hypothesized, our depression intervention did produce reductions in bulimic symptoms, although these effects did not persist for the full 6-month follow-up period. It was noteworthy that the intervention effects were not solely driven because the intervention impacted the cognitive symptoms of bulimia nervosa, as the intervention effects remained when the items assessing overvaluation of weight and shape were omitted from the bulimic symptom composite. In addition, mediation analyses provided support for the hypothesis that change in depressive symptoms mediated the effect of the intervention on change in bulimic symptoms. However, it is important to consider the possibility that the intervention effects for bulimic symptoms are solely a product of non-specific factors, such as participant expectancies, demand characteristics, or therapeutic attention. Two considerations argue against this alternative explanation for this effect. First, these non-specific factors would increase the likelihood of observing effects for all outcome variables, but we did not observe effects for substance use in the present trial. If these non-specific factors were sufficient to produce effects, they should have done so for all outcomes. Second, because the program was solely described as an intervention to reduce current and future depression and because participants were kept blind to the hypothesis that this intervention might reduce bulimic symptoms, it seems unlikely that expectancies or demand characteristics produced the intervention effects for bulimic symptoms.

To our knowledge, these results provide the first experimental evidence from a prevention trial that decreasing depressive symptoms may reduce bulimic pathology. These findings echo the evidence from double-blind randomized treatment trials that suggest that antidepressant medications result in significant decreases in bulimic symptoms among adults meeting full diagnostic criteria for this eating disorder (47). These findings also converge with the evidence that depressive symptoms predict onset of bulimic pathology (6,8,48) and may be cautiously interpreted as evidence that depression promotes bulimic symptoms.

Interestingly, the fact that bulimic symptoms also predict future increases in depressive pathology (8,49), and that treatments for bulimic symptoms produce reductions in depressive symptoms (50-51), suggests that depression and bulimic pathology are likely reciprocally related, as has been previously theorized (52). It was noteworthy that a depression intervention that did not include content targeting eating or bulimic symptoms produced decreases in bulimic symptoms. However, the fact that these findings faded over the follow-up period suggests that interventions solely targeting depression may not be sufficient for preventing or reducing bulimic pathology. Nonetheless, it may be worthwhile for future eating disorder prevention programs to include a component focusing on the reduction of depressive symptoms.

In contrast to expectations, our results indicated that despite significant reductions in depressive symptoms among intervention participants, there were no concomitant decreases in substance use. These findings offer little support for the assertion that substance use is linked to affective disturbances, as suggested by the negative affect theory. Our null findings converge with those from prospective studies that have found non-significant or weak relations between depressive symptoms and future increases in substance use (22-24). Although the correlation between depression and substance use is established, there may be other explanations for this relation. For instance, some omitted third variable may explain this relation. One possibility is that parental substance abuse increases risk for offspring substance use because of genetically convened risk (e.g., deficits in executive control) and also increases risk for depression because of the negative life events that often accompany parental substance abuse (53-54). In contrast, the correlation between depression and substance use may arise because the latter increases risk for the former. In support of this conjecture, several prospective studies have found that substance use predicted future increases in depression (22,55). In theory, regular use of psychoactive substances leads to neurological changes that result in attenuated positive affect and the use-related negative consequences of substance use may create dysphoric mood (56). Future research should examine this alternative relation between these variables. However, it is also possible that our sole focus on females may have impacted our findings. Because boys and men typically report elevated levels of substance use relative to girls and women (57-58), the former may be more likely than the latter to use psychoactive substances to cope with negative affect or to experience affective disturbances as a result of prolonged substance use. It is also possible that the depression intervention might have reduced substance abuse or dependency symptoms, which were not assessed in this study. It would be useful if future research examined these possibilities.

The relatively severe level of baseline depressive symptoms among sample participants deserves comment. Other adolescent depression prevention programs have reported lower baseline mean BDI scores (17-18.4) among “high risk” participants (e.g., (59-60)), although our mean values are closer to those observed in many controlled bulimia treatment studies, with mean BDI scores in the range of 22.9-25.5 (61-63). It is likely that the higher mean BDI values observed for the present sample occurred because we did not use diagnostic interviews to exclude participants with current major depression at baseline, which was the approach taken in most prior depression prevention trials. The present findings should be interpreted with this factor in mind.

It is important to consider the limitations of this trial when interpreting these findings. First, we relied solely on self-report questionnaires to assess changes in the outcomes. Unfortunately, limited resources prevented us from obtaining structured diagnostic interview data on the participants in this preliminary study. Although any limitations inherent to questionnaires should operate equally in our intervention and control conditions, self-report measures are generally lower in specificity than structured diagnostic interviews. Second, since we focused solely on females in this study, these findings may not generalize to males, especially in light of the gender differences in eating disorder diagnosis and substance use frequency. It would be useful if future research replicated this trial with male participants. Lastly, although the use of a waitlist control condition facilitated comparisons to the results obtained in prior CBT interventions for depression, the use of a placebo control condition would have allowed stronger inferences about intervention effects. Specifically, it would have been possible to rule out the possibility that demand characteristics, expectancy effects, or attention artificially inflated estimates of intervention effects on depression reduction. A waitlist control group only allows investigators to rule out regression to the mean and measurement artifact alternative explanations for intervention effects. However, participants were blinded to our hypothesis that our depression intervention might reduce bulimic symptoms and substance use, which renders it unlikely that our effects are due to demand characteristics. In addition, the fact that we did not find significant effects for substance use outcomes also suggests that our findings for bulimic symptoms are unlikely rooted in expectancy or demand characteristic effects, as they should have impacted all outcomes. Notwithstanding, future research could improve upon our trial by incorporating diagnostic interviews and a placebo control condition with a mixed gender sample.

In sum, our intervention effectively reduced depressive symptoms over a six-month follow-up, which provided an opportunity to experimentally investigate the effects of long-term changes in depressive symptoms on other outcomes. Compared to the waitlist control condition, the intervention produced short-term reductions in bulimic symptoms, but not substance use, providing support for the affect-regulation theory of bulimic pathology, but little support for the notion that substance use is rooted in affective disturbances. We believe that this randomized trial provided a rigorous test of affect-regulation theories. The fact that the results from this experimental trial largely agree with the effects observed in prospective studies allows us to place even greater confidence in the assertion that affective disturbances play an important role in promoting bulimic symptoms. We feel it would be useful if future researchers used experimental trials that manipulate putative risk factors for psychiatric disturbances in an attempt to triangulate the findings from prospective risk factor studies.

Acknowledgements

This study was supported by a research grant (4396) from the Hogg Foundation at the University of Texas, as well as a National Research Service Award (MH64254), a Career Award (MH01708), and a research grant (MH67183) from the National Institute of Mental Health.

Thanks go to the many project research assistants, Courtney Byrd, Amy Folmer, Cassie Goodin, and Jacob Mase, a multitude of undergraduate volunteers, the Austin Independent School District, and the participants who made this study possible.

References

1. Leon GR, Fulkerson JA, Perry CL, Early-Zald MB. Prospective analysis of personality and behavioral vulnerabilities and gender influences in the later development of disordered eating. J Abnorm Psychol 1995;104:140–149. [PubMed: 7897036]
2. McCarthy M. The thin ideal, depression, and eating disorders in women. Behav Res Ther 1990;28:205–218. [PubMed: 2196049]

3. Cooley E, Toray T. Body image and personality predictors of eating disorder symptoms during the college years. Int J Eat Disord 2001;30:28–36. [PubMed: 11439406]
4. Stice E. A prospective test of the dual pathway model of bulimic pathology: Mediating effects of dieting and negative affect. J Abnorm Psychol 2001;110:124–135. [PubMed: 11261386]
5. Stice E, Presnell K, Spangler D. Risk factors for binge eating onset: A prospective investigation. Health Psychol 2002;21:131–138. [PubMed: 11950103]
6. Killen JD, Taylor CB, Hayward C, Haydel KF, Wilson DM, Hammer L, et al. Weight concerns influence the development of eating disorders: A 4-year prospective study. J Consult Clin Psychol 1996;64:936–940. [PubMed: 8916622]
7. Stice E, Agras WS. Predicting onset and cessation of bulimic behaviors during adolescence: A longitudinal grouping analysis. Behav Ther 1998;29:257–276.
8. Stice E, Burton E, Shaw H. Prospective relations between bulimic pathology, depression, and substance abuse: Unpacking comorbidity in adolescent girls. J Consult Clin Psychol 2004;72:62–71. [PubMed: 14756615]
9. Johnson JG, Cohen P, Kasen S, Brook JS. Eating disorders during adolescence and the risk for physical and mental disorders during early adulthood. Arch Gen Psychiatry 2002;59:545–552. [PubMed: 12044197]
10. Keel PK, Fulkerson JA, Leon GR. Disordered eating precursors in pre- and early adolescent girls and boys. J Youth Adolesc 1997;26:203–216.
11. Vogeltanz-Holm ND, Wonderlich SA, Lewis BA, Wilsnack SC, Harris TR, Wilsnack RW, Kristjanson AF. Longitudinal predictors of binge eating, intense dieting, and weight concerns in a national sample of women. Behav Ther 2000;31:221–235.
12. Cooper ML, Frone MR, Russell M, Mudar P. Drinking to regulate positive and negative emotions: A motivational model of alcohol use. J Pers Soc Psychol 1995;69:990–1005. [PubMed: 7473043]
13. McCollam JB, Burish TG, Maisto SA, Sobell MB. Alcohol's effects on physiological arousal and self-reported affect and sensations. J Abnorm Psychol 1980;89:224–233. [PubMed: 7365134]
14. Newcomb, MD.; Bentler, PM. Sage; Newbury Park, CA: 1988. Consequences of adolescent drug use: Impact on the lives of young adults.
15. Sher, KJ. Psychological characteristics of children of alcoholics: Overview of research methods and findings. In: Galanter, M.; Begleiter, H., editors. Recent developments in alcoholism, Vol 9: Children of alcoholics. Plenum Press; New York: 1991. p. 301-326.
16. Hankin BL, Abramson LY, Moffitt TE, Silva PA, McGee R, Angell KE. Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. J Abnorm Psychol 1998;107:128–140. [PubMed: 9505045]
17. Brook JS, Whitman M, Gordon AS. Stages of drug abuse in adolescence: Personality, peer, and family correlates. Dev Psychol 1983;19:269–277.
18. Kandel DB, Kessler RC, Margulies RZ. Antecedents of adolescent initiation into stages of drug use: A developmental analysis. J Youth Adolesc 1978;7:13–40.
19. Pardini D, Lochman J, Wells K. Negative emotions and alcohol use initiation in high-risk boys: The moderating effect of good inhibitory control. J Abnorm Child Psychol 2004;32:505–518. [PubMed: 15500030]
20. Tschann JM, Adler NE, Irwin CE. Initiation of substance use in early adolescence: The roles of pubertal timing and emotional distress. Health Psychol 1994;13:326–333. [PubMed: 7957011]
21. Stice E, Myers M, Brown S. A longitudinal grouping analysis of adolescent substance use escalation and de-escalation. Psychol Addict Behav 1998;12:14–27.
22. Brook JS, Cohen P, Brook DW. Longitudinal study of co-occurring psychiatric disorders and substance use. J Am Acad Child Adolesc Psychiatry 1998;37:322–330. [PubMed: 9519638]
23. Galaif ER, Sussman S, Chou C, Wills TA. Longitudinal relations among depression, stress, and coping in high risk youth. J Youth Adolesc 2003;32:243–258.
24. Stice E, Barrera M Jr. Chassin L. Prospective differential prediction of adolescent alcohol use and problem use: Examining mechanisms of effect. J Abnorm Psychol 1998;107:616–628. [PubMed: 9830249]

25. Stice E. Risk and maintenance factors for eating pathology: A meta-analytic review. Psychol Bull 2002;128:825–848. [PubMed: 12206196]
26. Glantz MD. Introduction to the special issue on the impact of childhood psychopathology interventions on subsequent substance abuse: Pieces of the puzzle. J Consult Clin Psychol 2002;70:1203–1206. [PubMed: 12472297]
27. Hinshaw SP. Intervention research, theoretical mechanisms, and causal processes related to externalizing behavior patterns. Dev Psychopathol 2002;14:789–818. [PubMed: 12549704]
28. Stice E, Mazotti L, Weibel D, Agras WS. Dissonance prevention program decreases thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: A preliminary experiment. Int J Eat Disord 2000;27:206–217. [PubMed: 10657894]
29. Brent DA, Holder D, Kolko DJ, Birmaher B, Baugher M, Roth C, et al. A clinical psychotherapy trial for adolescent depression comparing cognitive, family, and supportive therapy. Arch Gen Psychiatry 1997;54:877–885. [PubMed: 9294380]
30. Clarke G, Hawkins W, Murphy M, Sheeber L, Lewinsohn PM, Seeley JR. Targeted prevention of unipolar depressive disorder in an at-risk sample of high school adolescents: A randomized trial of group cognitive intervention. J Am Acad Child Adolesc Psychiatry 1995;34:312–321. [PubMed: 7896672]
31. Rehm LP, Kaslow NJ, Rabin AS. Cognitive and behavioral targets in a self-control therapy program for depression. J Consult Clin Psychol 1987;55:60–67. [PubMed: 3571660]
32. Addis ME, Jacobson NS. Reasons for depression and the process and outcome of cognitive-behavioral psychotherapies. J Consult Clin Psychol 1996;64:1317–1424.
33. Jacobson N, Dobson K, Traux P, Addis M, Koerner K, Gollan J, Gortner E, Prince S. A component analysis of cognitive-behavioral treatment for depression. J Consult Clin Psychol 1996;64:295–304. [PubMed: 8871414]
34. Clarke GN, Hornbrook M, Lynch F, Polen M, Gale J, Beardslee W, O'Connor E, Seeley J. A randomized trial of a group cognitive intervention for preventing depression in adolescent offspring of depressed parents. Arch Gen Psychiatry 2001;58:1127–1134. [PubMed: 11735841]
35. Stice E, Shaw H. Eating disorder prevention programs: A meta-analytic review. Psychol Bull 2004;130:206–227. [PubMed: 14979770]
36. Wilson, GT.; Becker, CB.; Heffernan, K. Eating Disorders. In: Mash, EJ.; Barkley, RA., editors. Child Psychopathology. 2nd. New York: Guilford: 2003. p. 687-715.
37. Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Applied Psychological Measurement 1977;1:385–401.
38. Beck AT, Steer RM, Garbin M. Psychometric properties of the Beck Depression Inventory: 25 years of evaluation. Clin Psychol Rev 1988;8:77–100.
39. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry 1961;4:561–571. [PubMed: 13688369]
40. Fairburn CG, Beglin SJ. Assessment of eating disorders: Interview or self-report questionnaire? Int J Eat Disord 1994;16:363–370. [PubMed: 7866415]
41. Fairburn, CG.; Cooper, Z. The eating disorder examination (12th edition). In: Fairburn, C.; Wilson, G., editors. Binge eating: Nature, assessment, and treatment. New York: Guilford: 1993. p. 317-360.
42. Black CM, Wilson GT. Assessment of eating disorders: Interview versus questionnaire. Int J Eat Disord 1996;20:43–50. [PubMed: 8807351]
43. Johnston, L.; O'Malley, P.; Bachman, J. National Institute on Drug Abuse, US Government Printing Office; Washington DC: 1988. Illicit drug use, smoking and drinking by America's high school students, college students, and young adults 1975-1987.
44. Schafer JL, Graham JW. Missing data: Our view of the state of the art. Psychol Methods 2002;7:147–177. [PubMed: 12090408]
45. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. J Pers Soc Psychol 1986;51:1173–1182. [PubMed: 3806354]
46. Clingempeel WG, Henggeler SW. Randomized clinical trials, developmental theory, and antisocial youth: Guidelines for research. Dev Psychopathol 2002;14:695–711. [PubMed: 12549700]

47. Zhu AJ, Walsh BT. Pharmacological treatment of eating disorders. Can J Psychiatry 2002;47:227–234. [PubMed: 11987473]
48. Johnson JG, Cohen P, Kotler L, Kasen S, Brook JS. Psychiatric disorders associated with risk for development of eating disorders during adolescence and early adulthood. J Consult Clin Psychol 2002;70:1119–1128. [PubMed: 12362962]
49. Stice E, Hayward C, Cameron R, Killen JD, Taylor CB. Body image and eating related factors predict onset of depression in female adolescents: A longitudinal study. J Abnorm Psychol 2000;109:438–444. [PubMed: 11016113]
50. Bailer U, de Zwann M, Leisch F, Strnad A, Lennkh-Wolfsberg C, El-Giamal N, et al. Guided self-help versus cognitive-behavioral group therapy in the treatment of bulimia nervosa. Int J Eat Disord 2004;35:522–537. [PubMed: 15101068]
51. Lowe MR, Davis WN, Annunziato RA, Lucks DL. Inpatient treatment for eating disorders: Outcome at discharge and 3-month follow-up. Eat Behav 2003;4:385–397. [PubMed: 15000964]
52. Heatherton, TF.; Polivy, J. Chronic dieting and eating disorders: A spiral model. In: Crowther, JH.; Tennenbaum, DL.; Hobfold, SE.; Parris, MA., editors. The etiology of bulimia nervosa: The individual and familial context. Hemisphere; Washington DC: 1992.
53. Chassin L, Pitts SC, Prost J. Binge drinking trajectories from adolescence to emerging adulthood in a high-risk sample: Predictors and substance abuse outcomes. J Consult Clin Psychol 2002;70:67–78. [PubMed: 11860058]
54. Nigg JT, Glass JM, Wong MM, Poon E, Jester JM, Fitzgerald HE, et al. Neuropsychological executive functioning in children at elevated risk for alcoholism: Finding in early adolescence. J Abnorm Psychol 2004;113:302–314. [PubMed: 15122950]
55. Fergusson DM, Lynskey MT, Horwood LJ. The short-term consequences of early onset cannabis use. J Abnorm Child Psychol 1996;24:499–512. [PubMed: 8886945]
56. Schuckit MA. Alcohol and depression: A clinical perspective. Acta Psychiatr Scand 1994;377s:28–32.
57. Bauman A, Phongsavan P. Epidemiology of substance use in adolescence: Prevalence, trends and policy implications. Drug Alchohol Depend 1999;55:197–207.
58. National Institute on Drug Abuse (NIDA). Sixth triennial report to the U.S. Congress on drug abuse and addiction research. US Government Printing Office; Washington, D.C.: 1999. DHHS Publication no. (ADM) 87-1519
59. Peden AR, Rayens MK, Hall LA, Beebe LH. Preventing depression in high risk college women: A report of an 18-month follow-up. J Am Coll Health 2002;49:299–306. [PubMed: 11413947]
60. Spence SH, Sheffield JK, Donovan CL. Preventing adolescent depression: An evaluation of the problem solving for life program. J Consult Clin Psychol 2003;71:3–13. [PubMed: 12602420]
61. Fairburn CG, Jones R, Peveler RC, Hope RA, O'Conner M. Psychotherapy and bulimia nervosa: Longer-term effects of interpersonal psychotherapy, behavioral therapy, and cognitive behavior therapy. Arch Gen Psychiatry 1993;50:419–428. [PubMed: 8498876]
62. Safer DL, Telch CF, Agras WS. Dialectical behavior therapy for bulimia nervosa. Am J Psychiatry 2001;158:632–634. [PubMed: 11282700]
63. Thackwray DE, Smith MC, Bodfish JW, Meyers AW. A comparison of behavioral and cognitive-behavioral interventions for bulimia nervosa. J Consult Clin Psychol 1993;61:639–645. [PubMed: 8370859]

Table 1

Means and Standard Deviations for the Cognitive Behavioral Therapy (CBT) Condition and Waitlist Control Condition on the Depressive Symptom, Bulimic Symptoms, and Substance Use scales and Results from the Paired Comparisons

	Baseline (Week 1) M (SD)	Termination (Week 4) M (SD)	1-month follow-up (Week 8) M (SD)	6-month follow-up (Week 20) M (SD)
Dependent variable				
Beck Depression Inventory				
CBT (n=74)	27.78 (9.85) $_a$	18.30 (10.72) $_b$	18.39 (11.21) $_b$	15.92 (11.54) $_b$
Waitlist (n=74)	24.13 (10.43) $_a$	23.81 (10.62)	19.57 (11.54) $_b$	16.64 (13.96) $_b$
Bulimic symptoms				
CBT (n=74)	26.40 (17.61) $_a$	20.28 (15.45) $_b$	18.30 (15.07) $_b$	19.60 (14.61) $_b$
Waitlist (n=74)	27.47 (17.36) $_a$	25.60 (18.13)	25.74 (18.31)	22.52 (14.27) $_b$
Substance use				
CBT (n=74)	11.63 (8.88)	11.01 (8.66)	11.20 (8.92)	10.86 (8.30)
Waitlist (n=74)	11.63 (8.73)	11.33 (8.94)	11.50 (9.18)	11.18 (8.58)

Notes: Means in the same row with different subscripts were statistically significantly different ($p < .016$).