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BRIEF REPORT

Efficacy Trial of a Selective Prevention Program Targeting Both Eating Disorders and Obesity Among Female College Students: 1- and 2-Year Follow-Up Effects

Eric Stice, Paul Rohde, Heather Shaw, and C. Nathan Marti
Oregon Research Institute, Eugene, Oregon

Objective: Evaluate the effects of a prevention program targeting both eating disorders and obesity at 1- and 2-year follow-ups. ***Method:*** Female college students at risk for these outcomes because of body image concerns ($N = 398$) were randomized to the *Healthy Weight 2* group-based 4-hr prevention program, which promotes lasting healthy improvements to dietary intake and physical activity and nutrition science health behavior change principles, or an educational brochure control condition. ***Results:*** Intervention participants showed significantly less body dissatisfaction and eating disorder symptoms and lower eating disorder onset through 2-year follow-up versus controls, but the former 2 effects were small. There were no main effects for body mass index (BMI), depressive symptoms, dieting, caloric intake, physical activity, or obesity onset. Moderator analyses revealed stronger eating disorder symptom effects for youths with initially elevated symptoms and lower pressure to be thin, stronger BMI effects for youths with initially elevated symptoms and BMI scores, and weaker eating disorder symptom effects for youths with initially elevated pressure to be thin. ***Conclusions:*** The 60% reduction in eating disorder onset over the 2-year follow-up was clinically significant and a novel effect for a prevention program, but the main effects on continuous outcomes were small, suggesting that adding nutrition science principles weakened the intervention efficacy. Effects on both eating disorder symptoms and BMI were greater for those with elevated eating disorder symptoms and BMI at pretest, implying that it might be useful to target these individuals in future trials.

Keywords: prevention, eating disorder symptoms, weight gain, body dissatisfaction, moderators

As eating disorders and obesity are both prevalent and associated with impairment, distress, morbidity, and mortality, it would be ideal to develop a prevention program that addressed both problems. To date, only two prevention programs have significantly improved both outcomes in a randomized trial.[1] The 3-hr *Healthy Weight* prevention program, which promotes participant-driven lasting healthy improvements to dietary intake and physical activity in young women with body image concerns, reduced eating disorder symptoms and body mass index (BMI) relative to assessment-only controls and alternative interventions through 3-year (yr) follow-up, reduced eating disorder onset relative to assessment-only controls through 3-yr follow-up, and reduced obesity onset relative to assessment-only controls and an alternative intervention at 1-yr follow-up and relative to assessment-only controls at 3-yr follow-up (Stice, Marti, Spoor, Presnell, & Shaw, 2008). The second is a refined 4-hr version of the *Healthy Weight* prevention program (*Healthy Weight 2*), which added nutrition science principles for making health behavior changes (e.g., replacing high-energy dense foods with low-energy dense foods), that produced significantly greater reductions in body dissatisfaction and eating disorder symptoms, and greater increases in physical activity, at posttest, and greater reductions in BMI and reported dieting at 6-month (mo) follow-up than controls who received a brochure regarding how to improve body image (Stice, Rohde, Shaw, & Marti, 2012). Moderator analyses revealed significantly greater reductions in eating disorder symptoms for those with initially elevated symptoms and pressure to be thin, and significantly greater reductions in BMI for those with initially elevated symptoms.

This report describes the effects of *Healthy Weight 2* at 1- and 2-yr follow-ups. Aim 1 was to test whether *Healthy Weight 2* participants showed greater reductions in eating disorder symp-

Eric Stice, Paul Rohde, Heather Shaw, and C. Nathan Marti, Oregon Research Institute, Eugene, Oregon.

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Correspondence concerning this article should be addressed to Eric Stice, Oregon Research Institute, 1715 Franklin Boulevard, Eugene, OR 97403. E-mail: estice@ori.org

[1] Technically, the dissonance-based eating disorder prevention program (the *Body Project*) also significantly reduced obesity onset at 1-year follow-up relative to both an assessment-only control condition and an alternative intervention (Stice, Shaw, Burton, & Wade, 2006), but this intervention was not designed to prevent unhealthy weight gain.

toms and BMI (primary outcomes) by 1- and 2-yr follow-ups, as well as a lower incidence of eating disorder and obesity onset during follow-up than controls. Aim 2 was to test for effects on secondary outcomes, including dietary intake, dietary restraint, physical activity, body dissatisfaction, and negative affect. Aim 3 was to test whether effects on the primary outcomes were stronger for youths with initial elevations in eating disorder symptoms and BMI, as they have more room for improvement, and weaker for youths with risk factors not addressed by the intervention, including depressive symptoms and perceived pressure for thinness.

Method

Participants were 398 young women (*M* age = 18.4 years, range = 17–20) with a mean BMI of 23.7 (*SD* = 4.3) at baseline who responded to recruitment mailings and flyers inviting women with body image concerns[2] to participate in a trial evaluating two body acceptance interventions. The sole exclusion criterion was a current diagnosis of anorexia nervosa (AN), bulimia nervosa (BN), or binge eating disorder (BED). Figure 1 provides information on participant flow. Participants provided interview and survey data at pretest, posttest, and at 6-mo, 1-yr, and 2-yr follow-ups. Stice et al. (2012) has provided details regarding the sample demographics, informed consent, random assignment,[3] content of the *Healthy Weight 2* intervention and the educational brochures, facilitator training and supervision, assessor training, quality assurance, and participant pay.

The Eating Disorder Diagnostic Interview assessed *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) eating disorder symptoms. Items assessing symptoms in the past month were summed.[4] This composite showed internal consistency (α = .89), inter-rater agreement (intraclass correlation coefficient [ICC] *r* = .93), and 1-week (wk) test–retest reliability (ICC *r* = .95) for 72 randomly selected participants in this trial, as well as sensitivity to detect intervention effects (Stice et al., 2008). We tested whether the intervention reduced onset of threshold/subthreshold AN, BN, and BED, as well as purging disorder among those free of these conditions at pretest following definitions used previously (Stice, Marti, Shaw, & Jaconis, 2009). Threshold and subthreshold eating disorder diagnoses have shown 1-wk test–retest reliability (κ = .96) and inter-rater agreement (κ = .86) and are associated with elevated mental health treatment, distress, and functional impairment (Stice et al., 2009). Directly measured BMI scores were used to reflect change in weight because these scores are superior to age- and sex-adjusted percentiles or BMI *z* scores for modeling change over time (Cole, Faith, Pietrobelli, & Heo, 2005). BMI correlates with dual-energy X-ray absorptiometry (DEXA) measured body fat (*r*s = .80–.90) and health measures, such as adverse lipoprotein profiles (Pietrobelli et al., 1998). We tested whether the intervention reduced risk for obesity onset (BMI > 30). The Block Food Frequency Questionnaire assessed intake of specific food types over the past 2 wks; it has shown 2-wk test–retest reliability (mean *r* = .69) and has correlated (*r* = .57) with 4-day food records (Block & Subar, 1992). The Dutch Restrained Eating Scale assesses various dieting behaviors; it has shown internal consistency (α = .95), 2-wk test–retest reliability (*r* = .82), and convergent validity with self-reported caloric intake (but not objectively measured caloric intake; van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986). The Paffenbarger Activity Questionnaire assessed exercise; it has shown 1-mo test–retest reliability (*r* = .72) and convergent validity with physical activity records (*r*s = .28–.86, Ainsworth, Leon, Richardson, Jacobs, & Paffenbarger, 1993). The Body Dissatisfaction Scale assessed discontent with body parts; it has shown internal consistency (α = .94), 3-wk test–retest reliability (*r* = .90), and predictive validity for eating disorder onset (Stice, Marti, & Durant, 2011). Items from the Schedule for Affective Disorders and Schizophrenia for School-Age Children interview assessed depressive symptoms: This composite has shown internal consistency (α = .75), 1-wk test–retest reliability (*r* = .93), and inter-rater agreement (*r* = .85; Stice, Rohde, Gau, & Wade, 2010). Participants reported perceived pressure to be thin from family, friends, dating partners, and the media on the Perceived Sociocultural Pressure Scale (Stice, Marti, & Durant, 2011); this scale has shown internal consistency (α = .88), 2-wk test–retest reliability (*r* = .93), and predictive validity for increases in eating disorder symptoms (Stice, Marti, & Durant, 2011).

Results

Retention was 96% at posttest, 94% at 6-mo and 1-yr follow-up, and 93% at 2-yr follow-up. *Healthy Weight 2* participants missed a total of 19.8% of the sessions, of which 7.3% were completed individually before the next session occurred (12.5% of the sessions were not made up). We fit unconditional mixed models with person nested within group using SAS 9.2 PROC MIXED using data from posttest, 6-mo, 1-yr, and 2-yr follow-ups as dependent variables. Pretest values of the outcome were used as a covariate. Restricted maximum likelihood was used to estimate variance for each level of the model, where Level 1 units were the measurement occasion, which were nested in the Level 2 unit (person), which were nested in the Level 3 unit (group). Wald *z* tests were used to test the significance of random effects. Variability in the person random intercept was significant for all outcomes, but variability in the group random intercept was not significant for any outcome, and was therefore eliminated from the models. Time was modeled as months since the intervention and was coded 0 at posttest. We included non-linear growth terms as necessary. A group-by-time interaction indicated that intervention effects differed across

[2] During the initial phone screen, potential participants were asked whether they had body image concerns (response options: *none*, *slight*, *moderate*, *severe*); we required a moderate or severe response for enrolment into this "body acceptance" trial. Although we did not require a particular score on a body dissatisfaction screening measure that has been associated with elevated risk for these public health problems, the mean body dissatisfaction score in the present sample at baseline (3.37, *SD* = 0.69) was half a standard deviation (0.52) above the mean score of similarly aged females from a community sample (*M* = 2.91, *SD* = 0.88; Stice et al., 2009), suggesting that we recruited a sample with elevated body dissatisfaction. Further, past studies have found that elevated body dissatisfaction increases risk for onset of both eating disorders and overweight (e.g., Haines, Neumark-Sztainer, Wall, & Story, 2007; Stice, Marti, & Durant, 2011).

[3] The research assistant responsible for random assignment was not involved in delivery of the prevention program.

[4] Items assessing the behavioral (e.g., frequency of binge eating episodes, vomiting, laxative/diuretic use, fasting, and excessive exercise) and attitudinal (e.g., fear of weight gain, overvaluation of weight/shape, feelings of depression or guilt about overeating) features of anorexia nervosa, bulimia nervosa, and binge eating disorder over the past month were summed to form the overall symptom composite. This symptom composite was normalized with a log base_{10} transformation, though raw items are reported in Table 2.

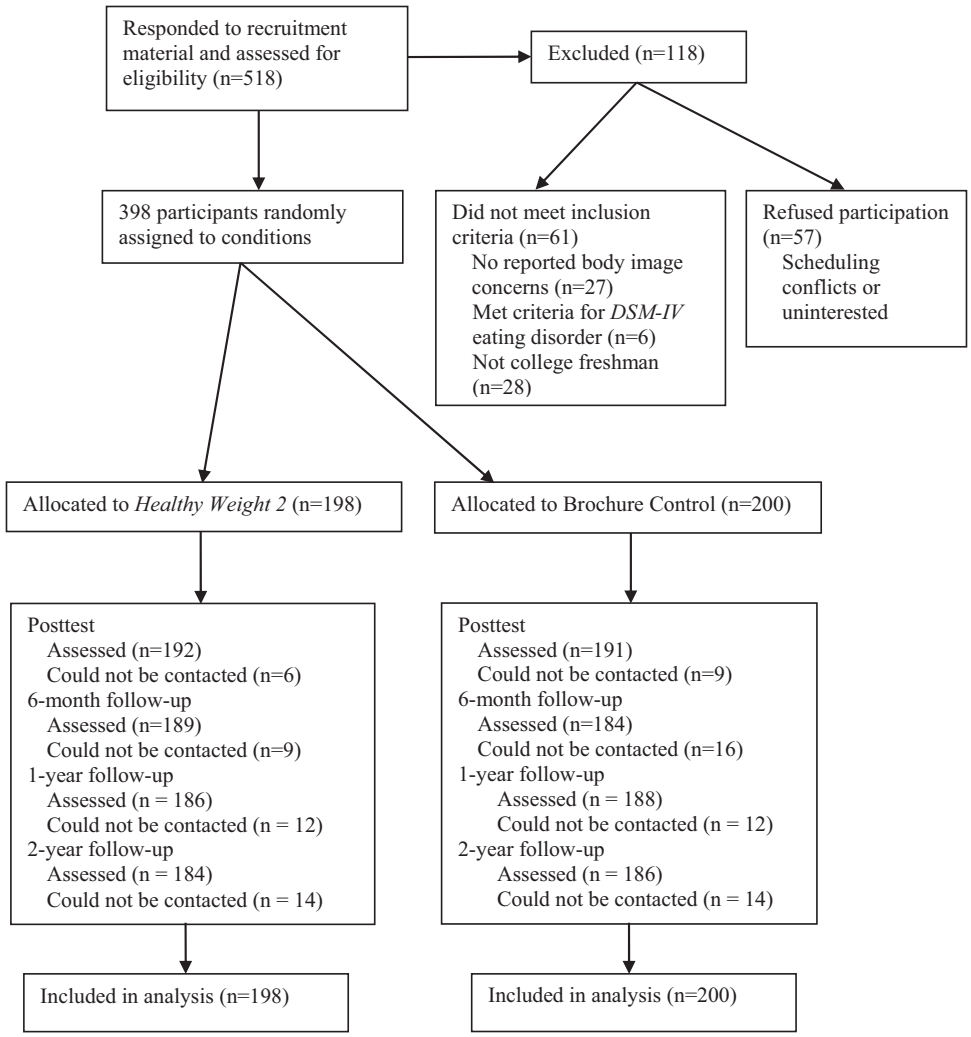

Figure 1. Participant flow throughout the study. *DSM-IV = Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994).

follow-up assessments, which were probed using simple slopes to compare groups at each time point, while holding other variables constant at their mean. Effect sizes were calculated by converting *t* values to *d* effect sizes. We used multiple imputations to account for missing data with the Amelia package of the R project. We imputed 20 data sets to reflect the errors that occur in real data, which were analyzed using PROC MIXED. Following the analysis of the 20 data sets, parameters and standard errors from the mixed models were combined using MIANALYZE to generate inferential statistics. We used an alpha of .05. There was a single case that exceeded the outlier criterion for the Cook's distance criterion; it was accounted for with an alternative intercept.

Groups did not differ on any pretest variables, including subthreshold eating disorders. Table 1 provides average means and standard deviations for the data sets. Intervention participants showed significantly lower eating disorder symptoms and body dissatisfaction ($d = -0.26$ and -0.22, respectively), but not BMI, depressive symptoms, or caloric intake, at 1- and 2-yr follow-up (see Tables 2 and 3). There were significant group and group-by-time effects in the physical activity model; intervention participants reported significantly greater physical activity than controls at posttest ($p = .013$, $d = 0.25$; which was reported in Stice et al., 2012), but not at other follow-ups.

Proportional hazard models implemented using SAS PROC PHREG tested whether onset of eating disorders or obesity differed across conditions. After excluding the 7% of the sample with a subthreshold eating disorder at pretest, 8.5% of controls and 3.4% of intervention participants showed eating disorder onset during the 2-yr follow-up, which was a significant difference ($\chi^2 = 3.12$, $p = .039$, hazard ratio [HR] = 2.24; see Figure 2).[5] Seven percent of the sample were obese at pretest and were excluded from the obesity onset

[5] Among the control participants, six exhibited subthreshold bulimia nervosa, one exhibited binge eating disorder, six exhibited subthreshold binge eating disorder, and two exhibited purging disorder. Among the intervention participants, three exhibited subthreshold bulimia nervosa, two exhibited subthreshold binge eating disorder, and one exhibited purging disorder.

Table 1
Means (and Standard Deviations) for Outcomes in the Control and Intervention Conditions

Variable	Condition	Pretest *M* (*SD*)	Postest *M* (*SD*)	6 months *M* (*SD*)	1 year *M* (*SD*)	2 years *M* (*SD*)
Primary						
Eating disorder symptoms	Control	10.44 (8.63)	8.27 (9.38)	6.43 (6.96)	6.73 (6.54)	6.98 (7.21)
	Intervention	10.22 (10.95)	6.11 (6.51)	6.05 (7.13)	5.90 (6.42)	5.22 (5.54)
BMI	Control	23.99 (4.48)	24.09 (4.51)	24.21 (4.56)	23.96 (4.56)	24.37 (4.94)
	Intervention	23.51 (4.14)	23.62 (4.14)	23.53 (4.13)	23.44 (4.06)	23.61 (4.22)
Secondary						
Body dissatisfaction	Control	3.37 (0.67)	3.23 (0.73)	3.04 (0.74)	3.02 (0.82)	3.02 (0.80)
	Intervention	3.37 (0.71)	3.09 (0.81)	2.97 (0.88)	2.94 (0.85)	2.88 (0.86)
Depressive symptoms	Control	1.40 (0.45)	1.15 (0.24)	1.21 (0.37)	1.23 (0.38)	1.35 (0.49)
	Intervention	1.43 (0.44)	1.12 (0.23)	1.21 (0.39)	1.29 (0.45)	1.32 (0.44)
Dieting	Control	2.98 (0.79)	2.87 (0.76)	2.74 (0.84)	2.68 (0.86)	2.64 (0.88)
	Intervention	2.88 (0.78)	2.69 (0.75)	2.54 (0.84)	2.49 (0.80)	2.43 (0.78)
Caloric intake	Control	1,357.14 (491.04)	1,285.47 (497.27)	1,300.90 (520.17)	1,335.60 (468.29)	1,274.87 (422.14)
	Intervention	1,344.39 (643.84)	1,205.83 (463.94)	1,239.40 (487.86)	1,268.82 (437.91)	1,285.53 (458.06)
Physical activity	Control	7,356.36 (6,437.07)	6,401.69 (5,759.14)	6,357.44 (5,627.12)	5,851.55 (4,624.36)	6,027.64 (6,286.30)
	Intervention	6,085.46 (4,203.74)	7,018.53 (4,566.51)	5,665.06 (4,587.22)	5,389.57 (4,946.04)	5,321.50 (4,475.40)

Note. Means and standard deviations are based upon averaged values across the 20 imputed data sets. Control $n = 200$; Intervention $n = 198$. BMI = body mass index.

analyses. There were no significant differences in obesity onset over follow-up ($\chi^2 = 0.74$, $p = .195$, HR = 1.72).

We conducted an analysis of reliable change scores (Jacobson & Truax, 1991) to evaluate the clinical significance of the main effects for eating disorder symptoms and body dissatisfaction. We used data from non-disordered participants in a previous study (Stice et al., 2009) to obtain standard deviations for a normal population for the reliable change index. Significantly more intervention versus control participants showed reliable *reductions* in body dissatisfaction over follow-up ($\chi^2 = 6.32$, $p = .012$), but there was no significant difference for eating disorder symptoms ($\chi^2 = 0.19$, $p = .665$). Marginally more controls versus intervention participants showed reliable *increases* in eating disorder symptoms over follow-up ($\chi^2 = 2.99$, $p = .084$), but there was no significant difference for body dissatisfaction ($\chi^2 = 0.32$, $p = .573$).

Moderator models included all variables in the main effects models and all two- and three-way interactions between group, time, and the moderator. We did not mean center the variables because group was dummy coded and time was centered at posttest so that the parameter estimates for these terms reflect the main effect of group and whether effects varied over follow-up.[6] The group-by-pretest eating disorder symptoms interaction was significant ($p = .013$, $d = 0.25$); for those at 1 *SD* above the pretest eating disorder symptoms mean, the intervention group showed significantly less eating disorder symptoms than controls over follow-up ($p = .005$, $d = 0.28$), but there were no intervention effects for those at 1 *SD* below the pretest mean. There was a significant group-by-time-by-pressure to be thin interaction

Table 2
Model Parameters for Primary Outcomes

Variable	Model component	Parameter	*B*	*SE*	*t*	*p*	*d*
Eating disorder symptoms	Fixed	Intercept	3.98	0.60	6.61	.000	0.66
		Pretest eating disorder symptoms	0.34	0.07	4.99	.000	0.50
		Quadratic pretest eating disorder symptoms	0.00	0.00	1.37	.171	0.14
		Intervention	−1.52	0.59	−2.57	.011	−0.26
		Natural log of month	−0.46	0.15	−3.05	.002	−0.31
		Intervention × Natural Log of Month	0.24	0.22	1.09	.277	0.11
	Random	Intercept	10.89				
		Month	0.23				
		Residual	23.71				
BMI	Fixed	Intercept	0.60	0.23	2.65	.008	0.27
		Pretest BMI	0.98	0.01	108.18	.000	10.85
		Intervention	−0.01	0.10	−0.05	.956	−0.01
		Natural log of month	0.05	0.04	1.11	.269	0.11
		Intervention × Natural Log of Month	−0.07	0.06	−1.13	.257	−0.11
	Random	Intercept	0.00				
		Month	0.19				
		Residual	0.97				

Note. BMI = body mass index.

Table 3
Model Parameters for Secondary Outcomes

Variable	Model component	Parameter	*B*	*SE*	*t*	*p*	*d*
Body dissatisfaction	Fixed	Intercept	0.45	0.12	3.87	.000	0.39
		Pretest body dissatisfaction	0.82	0.03	25.22	.000	2.53
		Intervention	−0.12	0.05	−2.23	.027	−0.22
		Natural log of month	−0.07	0.01	−4.73	.000	−0.47
		Intervention × Natural Log of Month	0.01	0.02	0.34	.733	0.03
	Random	Intercept	0.12				
		Month	0.01				
		Residual	0.17				
Depressive symptoms	Fixed	Intercept	0.68	0.04	17.61	.000	1.77
		Pretest depressive symptoms	0.32	0.02	13.38	.000	1.34
		Intervention	−0.03	0.03	−1.02	.306	−0.10
		Natural log of month	0.05	0.01	4.96	.000	0.50
		Intervention × Natural Log of Month	0.01	0.02	0.74	.458	0.07
	Random	Intercept	0.00				
		Month	0.01				
		Residual	0.08				
Dieting	Fixed	Intercept	0.74	0.09	7.92	.000	0.79
		Pretest dieting	0.71	0.03	24.93	.000	2.50
		Intervention	−0.11	0.05	−1.94	.053	−0.19
		Natural log of month	−0.07	0.02	−4.69	.000	−0.47
		Intervention × Natural Log of Month	−0.01	0.02	−0.34	.736	−0.03
	Random	Intercept	0.12				
		Month	0.01				
		Residual	0.18				
Caloric intake	Fixed	Intercept	396.11	75.14	5.27	.000	0.53
		Pretest BFFQ	0.75	0.08	9.43	.000	0.95
		Quadratic pretest BFFQ	−0.00	0.00	−3.21	.001	−0.32
		Intervention	−61.17	37.50	−1.63	.104	−0.16
		Natural log of month	7.93	19.07	0.42	.678	0.04
		Intervention × Natural Log of Month	9.32	26.57	0.35	.726	0.04
	Random	Intercept	37,456.09				
		Month	13,656.40				
		Residual	94,190.79				
Physical activity	Fixed	Intercept	3,089.11	364.31	8.48	.000	0.85
		Pretest Paffenbarger Activity Questionnaire	0.46	0.03	16.05	.000	1.61
		Intervention	1,064.52	424.00	2.51	.012	0.25
		Natural log of month	−147.76	124.94	−1.18	.238	−0.12
		Intervention × Natural Log of Month	−410.45	178.43	−2.30	.022	−0.23
		Intercept	3,756,682				
		Month	384,647.0				
		Residual	14,825,169				

Note. BFFQ = Block Food Frequency Questionnaire.

($p = .003$, $d = 0.30$) for eating disorder symptoms; intervention participants at 1 *SD* above the pretest pressure to be thin mean showed significantly lower eating disorder symptoms at posttest than controls ($p = .017$, $d = 0.24$), but the groups did not differ at the other follow-ups, whereas intervention participants at 1 *SD* below the pretest pressure to be thin mean did not differ from control participants at posttest, but showed significantly lower eating disorder symptoms at 6-mo ($p = .009$, $d = 0.27$), 1-yr ($p = .003$, $d = 0.30$), and 2-yr ($p = .002$, $d = 0.31$) follow-ups. The group-by-time-by-pretest eating disorder symptom interaction was significant ($p = .021$, $d = 0.23$) for BMI; for those at 1 *SD* above the pretest eating disorder symptoms mean, intervention versus control participants showed significantly lower BMI than controls at 6-mo ($p = .021$, $d = 0.23$), 1-yr ($p = .002$, $d = 0.31$), and 2-yr ($p < .001$, $d = 0.35$) follow-ups, whereas there were no significant effects for those at 1 *SD* below the pretest mean. The group-by-time-by-pretest BMI interaction was significant ($p = .022$, $d = 0.23$) for BMI; for those at 1 *SD* above the pretest BMI mean, intervention versus control participants showed significantly lower BMI at 6-mo ($p = .015$, $d = 0.24$), 1-yr ($p = .001$, $d = 0.33$), and 2-yr ($p < .001$, $d = 0.37$) follow-ups, but there were no effects for those at 1 *SD* below the pretest mean.

Discussion

Healthy Weight 2 participants showed significantly lower eating disorder symptoms at 1- and 2-yr follow-up, which is encouraging because only two prevention programs have produced effects for this outcome that persist through multiple-year follow-ups (Stice et al., 2008; Stice, Rohde, Shaw, & Gau, 2011). That this intervention

[6] Note that when we used mean-centered data for group, time, and the moderators in the models testing for moderation, the same interactive effects emerged.

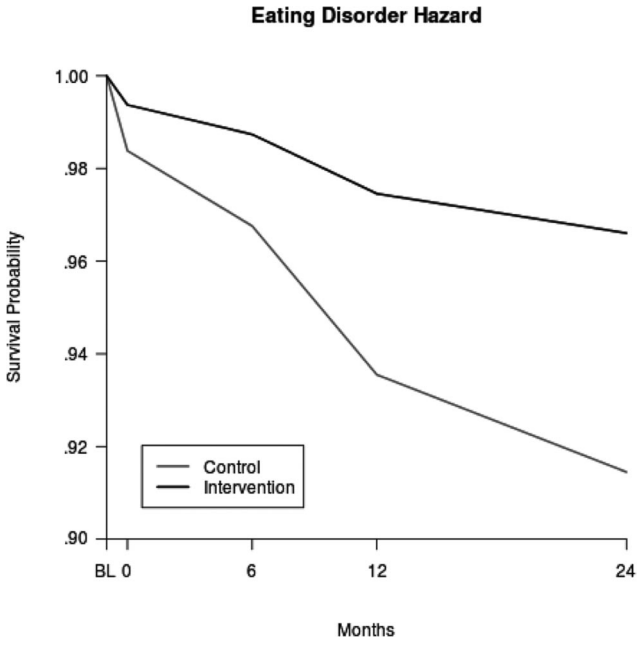

Figure 2. Cumulative hazard curves for eating disorders onset over 2-year follow-up in the *Healthy Weight 2* intervention condition and the educational brochure control condition. BL = baseline.

was only 4 hr in duration makes this effect even more striking. The effect size was relatively small ($d = -0.26$), and reliable change score analyses were only partially significant, though this effect was twice as large as the average effect size for reductions in eating disorder symptoms from eating disorder prevention trials with 1- and/or 2-yr follow-up assessments ($d = -0.13$).[7] This finding echoes the significant reduction in eating disorder symptoms observed at 3-yr follow-up for the original *Healthy Weight* intervention (Stice et al., 2008). Theoretically, the intervention helped participants develop healthier weight control strategies, which reduced reliance on transient dieting and other unhealthy weight control behaviors that characterize eating disorders. More importantly, *Healthy Weight 2* participants showed a 60% reduction in eating disorder onset over the 2-yr follow-up, which was a clinically significant difference that represented a large effect (HR = 2.2). This effect replicated the 60% reduction in eating disorder onset observed for the original *Healthy Weight* prevention program (Stice et al., 2008). This finding is novel, because only the original *Healthy Weight* prevention program and the dissonance-based *Body Project* have significantly reduced onset of blinded interview-assessed eating disorders over a multiyear follow-up.[8]

Healthy Weight 2 participants showed significantly lower BMI at 6-mo follow-up, but this effect was not significant at either 1- and 2-yr follow-up, and this intervention did not reduce obesity onset. It was encouraging that this prevention program produced effects for both eating disorder symptoms and BMI, as only two prevention programs have produced intervention effects for both of these outcomes (Stice et al., 2008, 2012), though a treatment intervention for overweight individuals who reported binge eating produced significantly greater weight loss for intervention versus control participants and may have reduced objective and subjective binge eating in intervention participants[9] (Jones et al., 2008). However, the fact that the original 3-hr *Healthy Weight* intervention produced larger and more persisting effects for BMI and risk for obesity onset (Stice et al., 2008) implies that adding principles to facilitate healthy dietary and exercise changes, largely drawn from nutrition science, attenuated intervention effects. The lack of such explicit guidelines in the original *Healthy Weight* intervention may have put more of the onus for change on participants, increasing personal investment and motivation to maintain the selected dietary and exercise changes. The inclusion of the dietary principles may also have complicated the simple message of the original intervention, which was that making small, lasting, incremental healthy changes to dietary intake and activity level could foster healthy weight control. This interpretation suggests that it might be useful to attempt to replicate the intervention effects on both eating disorder symptoms and BMI that were observed for the simpler original *Healthy Weight* prevention program.

In terms of secondary outcomes, the *Healthy Weight 2* intervention produced significantly lower body dissatisfaction through 1- and 2-yr follow-ups, echoing effects from the original *Healthy Weight* intervention that persisted through 1-, 2-, and 3-yr follow-ups. Perhaps the focus on the healthy-ideal versus the thin-ideal promoted body satisfaction. Attempting to make positive lifestyle changes may also improve body satisfaction. However, the effects for body dissatisfaction were relatively small, and the reliable change score analyses produced mixed support in this trial, and there were no long-term intervention effects on depressive symptoms, dieting, caloric intake, and physical activity. Presumably, the lack of effects for caloric intake and physical activity explain the absence of a longer-term effect on BMI.

Moderator analyses revealed that the effects on eating disorder symptoms at 1- and 2-yr follow-up were significantly larger for participants with initial elevations in eating disorder symptoms, extending results observed in the report focusing on the acute effects, which observed similarly stronger intervention effects at 6-mo follow-up (Stice et al., 2012). Presumably this is because the participants with elevated eating disorder symptoms scores at pretest had a greater opportunity to show reductions in this outcome. Moderator analyses also revealed that intervention effects on eating disorder symptoms varied as a function of pretest perceived pressure to be thin. As hypothesized, the intervention produced stronger effects over long-term follow-up for participants who reported lower versus higher pretest pressure to be thin. However, the fact that this effect was reversed at posttest suggests

[7] We calculated the effect sizes based on published trials using meta-analytic procedures.

[8] Although Taylor et al. (2006, p. 885) found "no overall significant differences in onset of eating disorders between intervention and control groups," moderator analyses indicated a significant reduction in eating disorder onset for the subset of overweight and obese participants (BMI > 25) but not among leaner participants, though they did not confirm all suspected cases with blinded diagnostic interviews.

[9] Jones et al. (2008) reported that Mann–Whitney testing indicated significant reductions in objective and subjective binge eating among intervention participants, but based on their article, they did not test whether the reduction in intervention participants was significantly greater than in control participants. Moreover, because the Mann–Whitney test was designed for independent rather than dependent samples, it would not be appropriate to use this procedure to test whether participants in the intervention condition showed reductions in objective and subjective binge eating over time.

that it might be best to focus on the subgroups that showed stronger intervention effects identified by the other moderators. Last, moderator analyses showed that the *Healthy Weight 2* intervention produced significantly lower BMI at 6-mo, 1-yr, and 2-yr follow-up for participants with pretest elevations in both eating disorder symptoms and BMI. These results are encouraging given the difficulty of obesity prevention and extend the evidence from the report focusing on the acute effects (Stice et al., 2012) in suggesting that the *Healthy Weight 2* intervention does significantly reduce BMI for participants with eating disordered behaviors. Presumably by reducing disordered eating behaviors (e.g., binge eating), participants are at a reduced risk for unhealthy weight gain. The evidence that the intervention also produced lower BMI for those with the highest initial BMI implies the intervention reduced excessive weight gain for those at greatest risk for future weight gain by virtue of initially elevated adiposity. These results, and those from the acute effects report, imply an intriguing interplay between eating disorder symptoms and unhealthy weight gain in young women with body image concerns. Collectively, the moderation effects suggest that it might be prudent to target young women who report a confluence of body image concerns, disordered eating behavior, and elevated initial BMI in future selective prevention trials that seek to reduce risk for both eating disorders and obesity.

Regarding limitations, the sample included college students who were largely White, suggesting that care should be taken in generalizing the results to more ethnically and educationally diverse populations that also suffer from eating disorders and obesity. The sample consisted solely of young women, implying that results should not be generalized to young men. Also, because self-report measures of dietary intake and physical activity are biased, it would have been preferable if objective measures of these outcomes had been used.

Results suggest that there may be value in refining this intervention to produce larger effects, such as by incorporating dissonance-induction procedures to promote greater reductions in energy dense food intake and greater increases in physical activity, though they also suggest it might be valuable to attempt to replicate the effects of the simpler *Healthy Weight* prevention program. The present findings also imply that it might be advantageous to target young women who have initial elevations in disordered eating behaviors and BMI in what might be best construed as indicated prevention programs (e.g., Jones et al., 2008). Given that both eating disorders and obesity are very prevalent, yet resistant to treatment, it will be vital to develop and disseminate prevention programs that effectively reduce these two major public health problems.

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